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CHEMICAL TESTING IN CASES OF "DRIVING UNDER THE INFLUENCE": A REVIEW OF THE LITERATURE.

By NORMAN E. W. MCCALLUM,
Department of Pathology, University of Melbourne.

In recent years the subject of chemical testing for the blood alcohol content in cases of "driving under the influence" has come into prominence; this review is an examination of part of the literature on ethyl alcohol in body fluids.

In 1951 the Victorian legislation dealing with the offence of "driving under the influence" was consolidated in the *Motor Car Act* of that year. Section 79 of this Act is in the following terms:

(1) (a) Every person driving a motor car who is apparently under the influence of intoxicating liquor or of any drug may be apprehended without warrant by any member of the police force and charged with an offence against this Act

(2) (a) Every person in charge of a motor car on a highway who is apparently under the influence of intoxicating liquor or of any drug may be apprehended

(c) For the purposes of this sub-section a person shall not be deemed to be in charge of a motor car unless he is attempting to start or drive the motor car or unless there are reasonable grounds for the belief that he intends to start or drive the motor car.

It will be seen that this offence is committed if a person who is "under the influence" of intoxicating liquor is actually engaged in driving a motor car, or even if he only attempts or intends to attempt to start or drive the car. There is no necessity that the car should, at any stage, be put in motion. What, then, is the legal test used to determine whether or not a person may be regarded as being "under the influence"?

In the South Australian case of *Pulleine v. Button* (1948. S.A.S.R. 1), Abbott J. made the following statement:

In my opinion, to find that a defendant is so much under the influence of intoxicating liquor as to be incapable of exercising effective control over his vehicle, means no more than to find beyond reasonable doubt that the defendant is so much affected by liquor as to be to a material degree incapable of exercising the same complete control in all respects over his vehicle—not merely in the driving of it—as a normal and reasonably competent driver would be able to exercise if he were entirely sober. In saying this, I will not, I hope, be understood to suggest that a defendant can be found guilty upon mere medical evidence that he has consumed

some liquor and is therefore, from a medical point of view, not strictly sober. The theories of some medical men about the effect in the blood stream of a minute quantity of alcohol have not yet attained to that degree of moral certainty upon which courts of law can act in the administration of the criminal law. It must always be a question of fact on all the evidence, upon which the court must be satisfied beyond reasonable doubt that the defendant is so much affected by liquor or drug that he is to a material degree not able to exercise the same complete control in all respects over his vehicle as a normal and reasonably competent driver would be able to exercise if he were sober.

However, it is to be noted that a person may be guilty of the offence of "driving under the influence" although, until the time when he was apprehended, there was nothing in the way in which he managed the vehicle to distinguish his driving from that of a sober driver. In the New South Wales case of *Molloy v. McDonald* (1939, 56 W.N. 159), Bavin J. gave the following opinion:

I have heard it said that there are persons who are quite as capable of driving and controlling a motor vehicle when they are drunk as when they are sober. Whether that is the fact or not, the legislature has paid no regard to it. It has not made incapacity for driving the test. The only test is whether the person driving is, in fact, under the influence of intoxicating liquor.

On the other hand, as Bavin J. pointed out, the question as to whether a man is capable of driving or controlling a motor vehicle may be a matter to be taken into account in considering whether he is under the influence of liquor. In the case of *Molloy v. McDonald* the evidence was that the defendant drove a motor lorry for some distance and then stopped and parked the vehicle. He drove and controlled it perfectly. The defendant left the parked lorry and walked unsteadily across the road. He was arrested and his breath smelt of liquor, his eyes were slightly bloodshot, and his speech was thick. The magistrate dismissed the charge on the ground that it was necessary to show the element of incapacity properly to drive and control the vehicle. The matter then came before Bavin J. by way of a case stated, and it was held that this element was unnecessary provided that it was shown that the defendant was under the influence of intoxicating liquor.

In Queensland a similar view was adopted in the case of *Noonon v. Elson* (1950, Q.S.R. 215), in which again it was held that proof of inability properly to control the vehicle was not required, it being sufficient for the prosecution to prove that, as a result of the consumption of liquor, the mental or physical faculties of the accused were so affected as to be no longer in a normal condition. In Western Australia, in *Washer v. Sullivan* (1948, W.A.L.R. 101), it was held that if evidence was confined entirely to the circumstances which pertained while the vehicle was in motion, there could be no successful prosecution until an accident had either occurred or had become so imminent as to be almost unavoidable. In this case Dwyer C.J. made the following statement:

I am of the opinion that no evidence of driving defaults is absolutely necessary, if the tribunal of trial is satisfied, from other circumstances, that the existing influence of liquor on the driver was sufficient to establish that he would have been incapacitated by that influence from exercising proper control in such circumstances as might be expected to occur at the place and time he was driving.

In the unreported Victorian case of *Rex v. Panluke*, the position was clearly stated by Low J.:

The Act does not require evidence that the defendant is drunk or that he is unable to drive the car; all that is necessary is to prove that he was under the influence of intoxicating liquor while driving the car.

In the light of the foregoing decisions it is not hyperbole to state that they do not greatly assist the medical practitioner in arriving at a basis or standard on which he may determine the condition of "under the influence". The medical examiner will not, in most cases, personally observe that an individual is incapable of driving his vehicle properly, so the foregoing decisions do not apply to his examination. However, there does not appear to be any reason why the medical examiner should not examine the subject from the standpoint of whether he is so influenced

by alcohol as to be incapable of controlling the vehicle properly. This view is expressed in the Western Australian case mentioned above. It would be in line with the English law on this offence, where the *Road Traffic Act 1930* includes the expression "under the influence of drink or a drug to such an extent as to be incapable of having proper control of the vehicle . . ." (Smith and Cook, 1948).

Once an opinion has been reached as to whether a person is or is not so influenced, this evidence is considered along with all the other evidence.

In most States of America legislation exists to authorize the admission as evidence of the results of chemical tests for alcohol.

The 1941 amendment to Section 70 of the *Motor Vehicle and Traffic Law* by the New York legislature (*Syracuse Law Review*, 1950, Volume 2) illustrates this. In this section three ranges are described: a concentration of 0.05% or less alcohol in the blood is *prima facie* evidence that the defendant is not in an intoxicated condition; evidence that there was a concentration between 0.05% and 0.15% is relevant, but is not to be given *prima facie* effect; evidence that there was a concentration of 0.15% or above may be admitted as *prima facie* evidence that the defendant was in an intoxicated condition.

Legislation such as this separates the first and third from the intermediate group, through which no clear lines are drawn. In the intermediate group (0.05% to 0.15% blood alcohol content) the evidence of the blood test would be regarded as one piece of evidence to be weighed and considered with the extrinsic evidence; it would not be given exaggerated importance in itself, though it is evident that the nearer the figure approached 0.05% or 0.15%, the greater would be its significance.

Concerning the legal point of the right of a medical practitioner to examine a person suspected of being "under the influence", the position here and in Britain is clearly defined in "Taylor's Principles and Practice of Medical Jurisprudence". According to this authority it is the duty of the practitioner to ask, in the presence of a witness, the consent of the suspected person before undertaking the examination; in addition the practitioner should inform the suspected person that he has the right to refuse to be examined, and that if the examination is performed the results may be given in evidence. It is also the duty of the medical practitioner to remind the person examined of his right to be further examined by a practitioner of his own choice. It is obvious that the same rules would apply to the taking of blood samples for chemical testing, and the suspected person should be offered a sealed sample of his own blood for independent testing.

Absorption.

Fairly complete investigation has been made into absorption of alcohol, its penetration into the tissues, and its eventual disappearance from the blood. One of the first major researches in this field was carried out in the United States in the nineteenth century by Grehan, who studied the penetration of alcohol into the tissues and constructed blood alcohol curves.

Shortly after administration, alcohol appears in all organs and secretions of the body. While food materials and water are absorbed only to a negligible extent through the gastric mucosa, alcohol is absorbed directly from the stomach (Best and Taylor, 1943). Diffusion experiments in which a human stomach, containing an alcohol solution, was immersed in normal saline and in fluid blood showed that rapid diffusion of alcohol occurred across the wall into the surrounding liquid (Bowden and McCallum, 1949).

Mellanby (1919) in animal experiments compared the effect on the blood alcohol concentration of a certain quantity of alcohol given in one dose with the effect when this same amount of alcohol was ingested in small quantities at varying time intervals. He found that when the alcohol was taken in one dose, the blood alcohol concentration reached its maximum in just under one hour and that the level was the same as that attained when the alcohol was divided into three portions taken at two

hourly intervals (the maximum concentration being produced about four and a half hours after the initial dose). He also observed that the rate of accumulation of alcohol in the blood increased (that is, the gradient of the pre-maximum part of the curve increases) with successive doses. He therefore concluded that the presence of alcohol in the blood facilitated the further absorption of alcohol from the alimentary canal.

It was also noted by Mellanby that at the end of the experiments, about six hours after alcohol was first given, the amount of alcohol in the blood in both cases was approximately the same. He therefore concluded that the rate of disappearance of alcohol from the body was constant and independent of the concentration. Haggard and Greenberg (1934) and Eggleton (1940) were unable to confirm this last conclusion; in fact, evidence pointed strongly to an increased rate of disappearance of alcohol from the blood with increasing concentration, and to the assumption that the rate of oxidation was proportional to the amount of alcohol present in the body.

A proportionality was found to exist by Mellanby between the blood alcohol concentration at its maximum and the amount consumed, unless the amount consumed was below two cubic centimetres per kilogram weight of his experimental animals, in which case the blood alcohol concentration became less than proportional.

Haggard, Greenberg and Rakieton (1940) gave a more comprehensive account of absorption when they discussed the principles governing the concentration of ethyl alcohol in the blood. As alcohol is absorbed from the alimentary canal into the portal circulation, the portal veins contain the highest concentration of alcohol in the body during the period of absorption. In the *vena cava* and the right side of the heart the concentration of alcohol is reduced by dilution with venous blood from other parts of the body. The alcohol concentration in arterial blood is always lower than in venous blood by the small amount of alcohol eliminated in expired air. The concentration of alcohol in the arterial blood supplying all organs is the same, but organs with a high blood supply receive a greater absolute quantity in a given time.

Haggard *et alii* (1940) investigated the concentrations at which respiratory failure occurred, and found that with adequate control the concentration of the jugular vein could serve as an index of alcohol tension in the respiratory centre.

The distribution, after equilibrium occurs, has been studied by numerous workers. Harger, Hulpieu and Lamb (1940) found that in animals, regardless of whether the alcohol was administered orally or by intravenous injection, equilibrium was reached after two to three hours. Eggleton administered alcohol to cats by intravenous injection and observed that a period of just under two hours from the time of the beginning of the injection was required for equilibrium to be attained in the whole body, although equilibrium between muscles and blood plasma was attained after thirty minutes.

Eggleton evaluated the relation between alcohol concentrations in the tissues and in the blood plasma and showed that after establishment of equilibrium the concentration of alcohol in all the tissues analysed was 70% to 80% of that of the blood plasma. The significant exception was a low value for fat, which was only 10% to 20% of the plasma concentration. These figures, when referred to blood instead of plasma, are in fair agreement with those of Harger and associates. A mathematical analysis of the curve of the concentration of alcohol appearing in the blood after oral administration was made by Haggard and Greenberg (1934). Evidence was produced which contradicted the conclusion of Mellanby that alcohol is oxidized at a uniform absolute rate, independent of concentration. These workers also disagreed with Mellanby concerning the completeness of absorption when the blood alcohol concentration reached its maximum. They found that the rate of oxidation was relative, not absolute—a feature which led them to recognize as a corollary that absorption was really a prolonged process.

In work performed on a dog, Haggard and Greenberg (1934) could not demonstrate this slow absorption experimentally, and it had to be derived mathematically. According to their calculations, some 40% of administered alcohol remains unabsorbed at the period of maximum concentration in the blood. Lack of agreement on the ratio of distribution of alcohol between blood and tissue may be an objection to this calculation. Recalculation of Eggleton's figures to apply to whole blood (on the basis of Miles's estimations in 1923 that the alcohol content of plasma is 10% to 20% higher than that of whole blood) shows that the ratio of Haggard and Greenberg is sufficiently accurate to make their contention valid. As the latter point out, even if they assume the ratio to be 1:1—that is, equal distribution between blood and tissue—the absorption is not complete at the maximum of the curve.

Continuing their analysis of the blood alcohol curve, Haggard and Greenberg explain that, in the first two hours, the concentration of alcohol rises in the blood because absorption exceeds oxidation, and at the summit of the curve (the plateau of Grehan) an approximate balance exists between absorption and oxidation. During the third to fifth hours the concentration declines at a nearly uniform absolute rate; but they found this rate to be less rapid than that which follows intravenous injection of alcohol. From this they conclude that absorption is still proceeding in the case of the orally administered alcohol. After the fifth or sixth hour the concentration ceases to decline at a uniform absolute rate and is now found to decline at a uniform percentage rate. At this stage they conclude that absorption from the alimentary canal is complete.

Metabolism, and Elimination.

Comprehensive studies of the equilibrium and post-maximum periods of blood alcohol level were carried out by Widmark (1922, 1933, 1934, 1935). Certain refinements of Widmark's work were supplied by subsequent studies, particularly that of Eggleton.

The quantity (a) of alcohol in grammes in the body when equilibrium is attained was given by Widmark in the equation

$$a = r p c \quad \dots \dots \dots (I)$$

where r is a constant (*der individuell Faktor*) and is the proportion of the body in alcohol equilibrium with the blood; p is the body weight in kilograms; and c is the concentration of alcohol in the blood in grammes per kilogram.

The value of r has been determined by some workers for their experimental animals, and has been given by Widmark (1932) for human subjects as 0.68 ± 0.085 for men, and 0.55 ± 0.055 for women. As has been indicated by Smith and Glaister, these differences of sex agree roughly with the concept of uniform distribution of alcohol in the tissues, excluding the fat and bones.

Others invest this constant with a somewhat lower value, and the minimal value is usually quoted over the range of 0.5 to 0.67. For accuracy it would be necessary to determine the value for each individual case. The value of the constant may be derived from the change in blood alcohol concentration with time. The graph in Figure I, taken from Eggleton (1940a), illustrates the calculation. The animal was given 4.95 grammes of alcohol intravenously. The straight line portion of the curve is extrapolated to zero time in the particular experiment, and this gives the theoretical initial concentration of alcohol in the plasma, C_0 , that is, the initial concentration if all the alcohol were immediately absorbed. Widmark has given for the total amount in grammes of alcohol absorbed the equation:

$$A = r p C_0 \quad \dots \dots \dots (II)$$

The value of A is here synonymous with the amount of alcohol given by injection, so that r may be calculated from the equation:

$$r = \frac{(\text{alcohol injected}), \text{ i.e., } A}{p C_0} \quad \dots \dots \dots (III)$$

According to Widmark's (1934) view, the presence of certain types of foodstuffs in the stomach is responsible for the "disappearance" of some of the ingested alcohol. Hence he adds the provision that if alcohol is administered orally, and the stomach contains no foodstuffs, then A , the amount absorbed, is approximately equal to the amount ingested. If the amount absorbed is less by some unknown quantity than the amount ingested, owing to such factors as suggested by Widmark, then this equation cannot apply.

Widmark observed that after the maximum alcohol concentration had been reached, the concentration diminished at a steady rate, although the actual amount disappearing in different individuals varied. Harger, Hulpien and Lamb (1937) in their experimental animals also observed a difference in the speed of removal of alcohol from the blood. However, they held the individual rate to be constant.

The metabolic rate, given in terms of Widmark's symbols, is thus:

$$\text{Metabolic rate} = r\beta$$

where β is held to be a constant, and is the rate of decrease of blood alcohol concentration which is determined experimentally. (Note that some workers refer

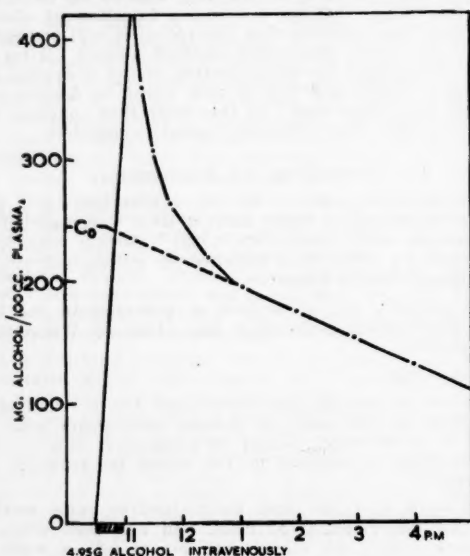


FIGURE 1.
Graph (Eggleton, 1940) showing plasma alcohol curve of cat weighing 3.26 kilograms. $C_0 = 2.35$ and $r = \frac{4.95}{3.26 \times 2.35} = 0.64$; $\beta = 0.0033$.

both factors r and β to plasma instead of to whole blood.) Haggard and Greenberg (1934) had found that, for dogs, the metabolic rate was directly proportional to the concentration. Eggleton raised a doubt concerning their technique on the grounds that, when r was calculated by Widmark's method for some of their experiments, it was found to have such values as 0.93 and 1.1. As she points out, this indicates that there is a concentration of alcohol in the whole body which is higher than that in the blood, which stands opposed to all previous work.

An ingenious experimental verification of the reliability of assessing the metabolic rate of alcohol from the values of r and β was provided by Eggleton (1940a). The rate was first calculated in the usual manner from the alcohol concentration curve, after which a constant infusion of alcohol was given for a long period at a rate approximately that of the calculated metabolic rate. From these experiments Eggleton demonstrated that the value of the metabolic rate obtained by the indirect assessment method was "a fair approximation" to the true value.

The rate of disappearance of alcohol from the blood (β), usually quoted as being within certain limits, has been known for some time to vary from person to person. The figure for humans has been quoted by Kerr (1939) at 10 millilitres of alcohol per hour, while Widmark records that alcohol disappears from the blood of human subjects at a value ranging from 4.1 to 11.1 grammes per hour (that is, from 5 to 14 millilitres per hour calculated as absolute alcohol). Smith and Glaister give the average value as 7.3 grammes per hour for males and 5.3 grammes per hour for females.

As the work of Widmark and Eggleton showed that determinations of metabolic rate were on a reliable basis, interest turned to a consideration of those factors which might be held to affect this rate. Those factors which affected the rate of absorption of alcohol were stated by Mellanby and others, but the mechanism by which these factors operate was not understood. It has been known for some time that certain individuals do not appear to be affected by a given amount of alcohol, while other individuals show definite signs indicating the consumption of alcohol. Rearrangement of a further equation of Widmark's, also quoted by Smith and Glaister in their "Recent Advances in Forensic Medicine", is as follows:

$$C_t = \frac{A}{pr} - t \quad \dots \dots \dots (iv)$$

where C_t is the concentration of alcohol in the blood t hours after its ingestion. This equation indicates that for a given amount of alcohol absorbed, A , the concentration in the blood after a certain time depends, among other constants, upon the body weight, p . This was put in another way by Mellanby, who pointed out that in the same experimental animals, whose body weight was allowed to vary, the inverse ratio of the alcohol concentration to the body weights was practically constant, regardless of whether the animals were fat or lean. However, it will be noted that equation (iv) does not imply proportionality between body weight and amount of alcohol in the blood.

When we are considering those factors which have been found to affect the rate at which alcohol is metabolized in the body, it will be remembered that the alcohol is distributed throughout all tissue, the fat and bone containing much less than other tissue. Loss of alcohol in urine and the breath contributes in a minor way towards the elimination. The disappearance has been demonstrated in the calorimetric studies of Atwater and Benedict (1902, cited by Mellanby, 1919) to occur almost completely by oxidation of alcohol in the body.

Foodstuffs.

The effect of foodstuffs on alcohol absorption has been investigated by several workers, including Mellanby, and later by Southgate (1925), by Leloir and Muñoz (1938), by Widmark (1934), and by Eggleton (1940b). Mellanby's controlled experiments showed that a food such as milk taken two to three hours prior to the alcohol not only reduced the maximum of blood alcohol concentration, but also reduced the rate of absorption from the gastrointestinal tract. Even the addition of small quantities of milk to the alcohol produced this inhibitory effect, although milk from which the fat had been removed was able to produce the effect only to a minor degree. These results led Mellanby to conclude that fats were responsible for this effect, and to predict that low melting point fats such as olive oil would produce more emphatic results—a prediction which Southgate in 1925 failed to substantiate. Widmark (1934) investigated a wider range of substances and observed two distinct effects produced by foodstuffs. First, there was a delayed absorption which resulted in a delayed and lower maximum concentration of alcohol in the blood; he attributed this to the metabolic action proceeding during the longer period of absorption. Secondly, he observed that a certain quantity of alcohol taken with or after the meal "disappeared" and was not metabolized; Southgate had already mentioned this fact. This effect, that certain food ingredients appeared to cause a proportion of ingested alcohol to vanish from the body, was called by Widmark the "specific effect of food ingredients".

It was found that the "specific effect" could be brought about by a number of foodstuffs, and was related to the protein content. Water, fat and carbohydrate were ineffective, but amino acids produced a strong and regular effect. Widmark postulated that the disappearance of the alcohol might be due to ester formation in the body; but Eggleton (1940b) later showed that such an hypothesis was unnecessary.

Clark and Morrissey (1938) differed from Widmark on the question of carbohydrate; they reported that the administration of insulin and glucose increased the rate at which alcohol was removed from the body, and suggested that the oxidation of alcohol might depend on simultaneous oxidation of glucose. Eggleton reinvestigated the problem using a method involving continuous intravenous infusion of alanine, as the effect of a single dose was too transitory for observation. She demonstrated that the metabolism of alcohol was increased by the addition of alanine, and that the hypothesis of disappearing alcohol was unnecessary. In her experiments on cats it was shown that r remained unaffected and that β , the rate of decrease of blood alcohol concentration, had increased. In Smith and Glaister's discussion on this subject it is not clear whether they regard β as constant in the one individual; however, this will be considered under the review of "tolerance".

The effect of the concentration of alcohol in the body has been studied by several workers, and it will already be clear from the discussion of Mellanby's work that he regarded the rate of decrease of blood alcohol concentration for a particular animal to be independent of the concentration in the blood. On the other hand, it has been generally accepted for some time that variation exists in the rate at which different individuals remove alcohol from their blood; hence the necessity to determine β experimentally. Haggard and Greenberg (1934) injected alcohol in suitable dilution into femoral veins of animals, and after the attainment of blood alcohol equilibrium they regarded the change in concentration of the alcohol in the arterial blood as a true criterion of change in the amount of alcohol in the body. They found that the alcohol level decreased at a nearly uniform rate each hour, and cited an average value of 17.6% per hour for the decrease. As has previously been pointed out, Eggleton threw doubt on their figure for metabolic rate when she drew attention to their very high values of r .

Newman, Lehman and Cutting (1937) experimented with dogs on the effect of concentration on the metabolic rate and found that each time the dose of alcohol was doubled there was an increase of 17% in the metabolic rate. Despite this finding, they state that "direct proportionality between blood alcohol concentration and rate of alcohol metabolism, claimed by Haggard and Greenberg, does not exist". In their curves of blood alcohol concentration, plotted against time, the "best fitting line" is linear in form, although curves of the form derived by Haggard and Greenberg could equally well, if not better, fit the points. Eggleton confirmed the fact that only very small variations in r could be detected, but found that in her animals (cats) the metabolic rate was directly dependent on the concentration of alcohol in the body. She found an increase of about 30% in metabolic rate for every 100 milligrammes per 100 millilitres increase in plasma alcohol concentration, and made the following statement:

This relationship, although apparent in every individual animal, could be demonstrated statistically on all the animals used only if the metabolic rate was expressed in terms of liver weight, rather than body weight or body surface.

The decline in the value of β with decreasing alcohol concentration in the plasma was found to be gradual, falling at such a rate as to appear constant over a period of two or three hours. Probably it is for this reason that most workers have considered the relation to be linear. On the other hand, it may be possible that the accelerated rate of removal of alcohol from the blood could be concealed by continuation of absorption of alcohol from the alimentary canal as suggested in the work of Haggard and Greenberg (1934). In her review of some of the factors which affected the metabolic rate of alcohol, Eggleton

included a series of experiments on the liver. Previous work had been conducted on the role of the pancreas in relation to alcohol oxidation (Clark and Morrissey, 1938; Mirsky and Nelson, 1939). By referring metabolic rate to liver weights of the experimental animals, Eggleton demonstrated a quantitative relationship between the metabolic rate and the alcohol concentration of the plasma. It was also observed that alcohol-accustomed animals were able to metabolize alcohol per unit of liver weight at a rate only 50% to 80% that of non-accustomed animals. A possible significance of this is discussed later.

There is some evidence that the liver is temporarily damaged during alcohol metabolism. Rosenthal (1930) observed that when a liver poison such as chloroform was given, animals returned to normal within two to seven days. However, when dogs were fed alcohol prior to chloroform, 60% died with acute yellow atrophy of the liver. The reverse of this procedure—feeding the alcohol after the chloroform—produced no greater liver damage than alcohol alone.

In recent years attention has been directed to dietetic deficiencies in chronic alcoholism, and it now seems to be generally accepted that the liver changes observed in the chronic alcoholic are due to these deficiencies. Alcohol consumption is thought to interfere with food intake. Whether this reduced food intake is associated with nausea or gastritis following alcohol consumption is not certain; but doubt exists among current workers as to whether the frequently reported gastritis due to alcohol has any basis in fact. Those interested in this aspect of liver damage will find the subject elaborated by Connor (1938) and by King and Perry (1947).

Tolerance.

The problem of tolerance to alcohol is one which has received a good deal of general comment in the literature; but in view of the importance of the matter in the medico-legal field it cannot be said to have received the attention it warrants. Tables have been prepared connecting the intoxication produced at various blood alcohol concentrations (Tables I and II). The usual objection to these tables is that they do not describe correctly the condition of those persons supposed to possess a high tolerance to alcohol. The principle is expressed by the observation of Newman and Fletcher (1940) that it is necessary to show that a certain blood alcohol concentration (150 milligrammes per 100 millilitres of blood) produces legal intoxication, not in the majority of individuals, but in all individuals. In view of objections such as this, the difficulty which has confronted those endeavouring to establish a general relation between alcohol concentration in a body fluid or tissue and the degree of intoxication in the individual is that, to include all cases, the maximum level must be raised to such a value that the cited concentration is of no medico-legal value. This is illustrated in the table of Muehlberger (1940, Table I).¹ Jetter (1938) examined 1000 individuals for acute alcoholic intoxication, and his table shows that of those with a blood alcohol concentration of 50 milligrammes per 100 cubic centimetres of blood, 10.5% were by his standards intoxicated; by the same standards he adjudged one individual not intoxicated who had a blood alcohol concentration of 0.40%. It may be noted here that Jetter is referring to intoxication, not to a state of being "under the influence"; equally relevant are the standards adopted for determining intoxication.

Reference to the subject of tolerance appears in the published works of Eggleton (1940a, 1940b), of Gaddum (1943), of Newman and Fletcher (1940), of Smith and Stewart (1932), and of others. One can only assume that they are discussing the same matter when they refer to tolerance, as there is seldom any attempt made to define exactly what they understand by "tolerance to alcohol". Gaddum, in a discussion of acetylcholine, points out that several different terms have been used to describe "what happens when the response of tissue to a drug is large at first, but diminishes or disappears although the drug is con-

¹ Report to the National Safety Council, U.S.A., 1940; no longer available in Melbourne.

tinued". Among these he includes tolerance. He states that the constant drinker suffers injury to the central nervous system and at the same time acquires a certain amount of tolerance "so that large quantities have no apparent effect on him". Gaddum also states that tolerance to alcohol is feeble when compared with the tolerance developed against morphine and cocaine, and continues as follows:

It [the tolerance] is mainly due to a real insensitivity of the central nervous system, and to the fact that the persistent drinker has much practice in concealing the effects of drink.

It will be gathered from the last statement that Gaddum regards tolerance as being due, in part, to habit, and as this same view arises later, it will be necessary to examine these two terms to assess any difference in their meaning.

Muehlberger, in his 1940 report to the Committee on Tests for Intoxication, United States of America, gave a fairly comprehensive account of the subject of tolerance. His definition appears to be included in the opening chapter:

It is a matter of common knowledge that persons vary widely in their response to alcohol. Some persons are affected more than others after drinking the same amount of intoxicating liquor. Those with better than average resistance are said to have a tolerance to alcohol.

Although this is not a satisfactory definition, his review of various types of tolerance should be examined in its light. He drew attention to four known types of tolerance—namely, consumption tolerance, constitutional or tissue tolerance, acquired tolerance and natural tolerance.

Consumption Tolerance.

The same amount of alcohol ingested does not produce in different individuals the same physiological response to the drug, nor does it produce the same alcohol concentration in the blood. Widmark refers to this as "consumption tolerance", and explains on this basis the different amounts of alcohol which different persons must consume to reach the same blood alcohol concentration. The committee referred to above took the view that at a given blood alcohol concentration there was no real difference in the degree of intoxication in different individuals, although it allows the differences of normal biological variation.

Constitutional or Tissue Tolerance.

Constitutional or tissue tolerance refers to the differences in response of different persons to the same concentration of alcohol in the blood. Biological variation is not included in constitutional tolerance, as the latter is meant, according to Muehlberger, to describe variations outside the limits of those biological variations which may vary by $\pm 10\%$ to $\pm 15\%$ from the norm.

Acquired Tolerance.

Acquired tolerance refers to the factor of habituation when repeated administration of alcohol over a period of time produces a progressively diminishing response to the drug.

Natural Tolerance.

Natural tolerance is a classification designed to include those persons who are total abstainers, or who have little recourse to alcohol, but in whom there is a very strong resistance to the effects of the drug. Persons who are reported to exhibit this tolerance may, although they seldom consume alcohol, drink comparatively large amounts without the production of responses commensurate with the amount consumed.

Comment.

Concerning consumption tolerance, Widmark, Mellanby, Southgate and others have shown that factors such as the presence of foodstuffs in the stomach operate to delay and diminish absorption of alcohol into the blood-stream. Such a phenomenon could hardly fall within the concept of tolerance. If equation (iv) is examined, it will be

seen that, for two individuals having absorbed the same amount of alcohol (a), the concentration of alcohol in the blood at time t (C_t) will depend upon the magnitude of the body weight (p), if we assume that the individual variations in β are small and may be neglected. However, the equation also shows that the body weight is connected in some way with the factor r —the proportion of the body in alcohol equilibrium with the blood. On first sight it seems from the equation that the lower the value of r , the greater will be the concentration of alcohol in the blood for a given amount of alcohol absorbed. The connexion between the factors p and r is difficult to determine, as little work has been carried out on this relationship. If a subject was to increase in weight over a period (that is, if p is increased), it may be assumed that this increase in body weight would for the most part be due to an increase in fat tissue, and Eggleton has found that fat in the body participates only to a small extent in the alcohol distribution. Consequently for this individual p has increased but r has decreased.

In an exhaustive alcohol study, Bahnson and Vedel-Petersen (1934) and Schmidt (1934) fixed their alcohol dosage for the experimental subjects (human) at 0.6 millilitre of absolute alcohol per kilogram of body weight. They observed variations among their subjects, both in the time taken to reach an alcohol maximum in the blood, and in the value of that maximum. If the foregoing reasoning is followed, it is probable that the administration of alcohol in amounts according to body weight may, in itself, be responsible for producing some of the variations observed, but, as the build of the subjects is not described, no information can be gained on this point.

Probably the most profitable manner of seeking a foundation for the concept of a tolerance is to consider the subject from these aspects: (i) the rate of absorption of alcohol from the alimentary canal; (ii) the rate of attainment of equilibrium in the body; (iii) the rate of disappearance of alcohol from the blood.

The experimental work on the rate of absorption from the alimentary canal has produced some conflicting results. Pringsheim (1908) concluded that with alcohol-habituated animals the absorption of alcohol was slightly slower than normal animals. However, subsequent work contradicted this—alcohol was absorbed more quickly in habituated animals—and the weight of evidence favours this view (Newman and Card, 1937). It is suggested (Cori, Villiaume and Cori, 1930) that the faster absorption is due to the accelerated passage of the contents of the stomach into the more permeable upper part of the intestine, and Newman and Card found on a quantitative scale that in habituated animals the food took only half the time to pass through the stomach that it took in normal animals. Consequently, one would not expect faster absorption from the alimentary canal to favour a tolerance. There is little information available concerning natural tolerance, and it is conceivable that natural tolerance and consumption tolerance are the same thing.

With regard to the attainment of alcohol equilibrium between blood and tissue, it would be expected that if some individuals showed a great lag in attaining this equilibrium there would be a higher concentration of alcohol circulating in the blood for a longer period. Harger *et alii* (1937) suggest that the higher alcohol concentration will not be confined to the blood, but may extend to those organs which have a better circulation, such as the brain. Gettler and Freireich (1931) state that it is the quantity of alcohol in the brain which determines the degree to which an individual is affected, so that variations among different persons in the attainment of alcohol equilibrium in the body may produce higher brain alcohol concentration in some. However, as there are no studies in the comparative rate of attainment of alcohol equilibrium in human subjects, any tolerance attributed to this factor must be speculative.

The third factor, the rate of disappearance of alcohol from the blood-stream, has already been discussed. It has been generally agreed that different individuals with the same alcohol concentration remove alcohol from the blood

at different rates, but the range of this variation is not great. The evidence is not satisfactory on the question of whether habituation to alcohol leads to an increase in the rate at which it can be removed from the blood. Pringsheim found that alcohol was removed more rapidly from the blood of tolerant animals than from normal animals, and this was attributed to increased alcohol metabolism, particularly in the liver. By using the technique of injecting alcohol into the blood, Newman and Cutting (1935) removed the interfering factor of absorption and they were unable to demonstrate any difference in the rate of metabolism in habituated animals.

The part played by the liver in alcohol tolerance has not been fully investigated. Eggleton (1940b) attempted to induce tolerance in cats by allowing free feeding of alcohol over a period of time, and subsequently interpreted the results obtained in terms of liver changes produced. The results did appear to show that alcohol feeding was accompanied by a small increase in liver weight. When the metabolic rates of these normal and tolerant animals were compared, no significant differences were detected if the results were expressed in terms of body weight. As has been mentioned, by referring these metabolic rates to liver weight, she found that the tolerant animals appeared to metabolize alcohol at 50% to 80% of the rate of the normal animals with the same liver size. The interpretation placed on these results by Eggleton was that they suggested an upper metabolic limit in the tolerant animal, and that this limit could not be exceeded by further increase of alcohol concentration in the blood. As Eggleton found that the metabolic rates, when referred to body weight, were not significantly different in normal and tolerant animals, it would be expected that two animals of the same body weight, one normal and one tolerant, would exhibit about the same metabolic rate. However, as the tolerant animal would be capable of metabolizing alcohol only at the rate of 50% to 80% of that of the normal animal, and as the increase in liver weight of the tolerant animal was observed to be small, a further suggestion presents itself that in the habituated animal some other tissue has assumed the function of alcohol oxidation. If this is so, there is no evidence to show that habituated animals can metabolize alcohol at any greater rate than abstaining animals.

From the review of the foregoing three factors nothing seems to have emerged connecting them directly with a tolerance. Muehlberger points out that it has been observed "by many careful workers, and upheld by the majority of valid evidence" that the extent of alcohol influence parallels the concentration of alcohol in the body fluids. This is to say, constitutional tolerance as such does not exist. In the lower range of blood alcohol concentrations the responses may be very dissimilar in different persons, depending, as McDougall (1932) points out, on their personality. Hyman (1939) has maintained that the differences are in the experimental subjects rather than in different actions of the drug; that is, the removal of inhibitions from a highly inhibited individual would produce a more spectacular change than it would from a person who was not ordinarily inhibited. The responses are all manifestations of the same stage of alcoholic influence—emotional instability. Muehlberger affirms that persons habituated to alcohol may be able to perform creditably purely physical and semi-automatic functions at much higher blood alcohol levels than can abstainers, but that this may be largely from habit rather than because of constitutional tolerance. In the higher concentration range, excellently described by McDougall, it seems to be agreed that there is no essential difference in different persons. This, together with the fact that most workers have placed the lethal dose of alcohol in the vicinity of 0.5%, regardless of whether the individual is or is not habituated to alcohol, raises another distinction when tolerance to such a drug as morphine is considered.

The fatal dose of morphine for a non-habituated person is about three grains ("Taylor's Principles and Practice of Medical Jurisprudence", Tenth Edition), but addicts may regularly take 75 grains or more per day without fatal results (Seever and Woods, 1953). This feature of

tolerance—the tremendous increase in fatal dose accompanying habituation—is one which appears to be absent in the case of alcohol, or, if present, is too small to be demonstrated. Hansman (1953) attributes to Goldberg the conclusion that heavy drinkers have an increased tolerance, but only "of the order of 0.04 gramme per centum of blood alcohol".

A critical review of the literature on the subject of tolerance is rendered difficult because there has been no standardization of the degrees of intoxication adopted by the various workers—indeed, it is doubtful if such standards could be selected. Jetter (1938) published a table in which one individual was adjudged "not intoxicated" with a blood alcohol concentration of 0.4%. Reference to this paper shows that his standards for intoxication demanded symptoms which another worker might require for an extremely advanced state of intoxication. In addition to this difficulty there is the inadequate distinction between tolerance and habit. If, as has been mentioned by Gaddum, the tolerance is due to a real insensitivity of the central nervous system and to the fact that the persistent drinker has much practice in concealing the effects of drink, then many will find themselves unable to reconcile this with their own conception of tolerance.

Alcohol in Blood.

On the subject of alcohol in blood and other body fluids, there has been speculation for some time as to whether the blood contains small amounts of ethyl alcohol as a naturally occurring constituent. Estimations have been made on "non-alcoholic" individuals to determine this point, and the most favoured figures of the numerous workers on "normal alcohol" prefer the range of 0.001% to 0.010%. The most convincing work in this respect was undertaken by Gettler, Niederl and Benedetti-Pichler (1932). In this work anhydrous ethyl alcohol was isolated from tissue in the following average amounts: human brain, 0.0004%; human liver, 0.0025%; human blood, 0.004%.

The relation between blood alcohol concentration and the degree of alcohol influence produced in the individual may be considered from two aspects: (i) by considering the effects produced within a certain range of blood alcohol concentration, and allowing for variations by "overlapping" the concentration ranges; (ii) by fixing ranges of blood alcohol concentrations and determining the percentage of persons exhibiting similar symptoms in the particular range.

TABLE I.

Muehlberger's Table (Prepared for the National Safety Council, United States of America).

1. Subclinical:	Blood: 0-120 mg./100 c.c., urine 0-160 mg./100 c.c. Normal by ordinary observation; slight change by special test.
2. Stimulation:	Blood 90-210 mg./100 c.c., urine 130-290 mg./100 c.c. Decreased inhibitions, emotional instability, slight incoordination, slowing of stimuli responses.
3. Confusion:	Blood 180-300 mg./100 c.c., urine 260-420 mg./100 c.c. Disturbance of sensation, decreased pain sense, staggering gait, slurred speech.
4. Stupor:	Blood 270-390 mg./100 c.c., urine 380-540 mg./100 c.c. Marked stimuli decrease, approaching paralysis.
5. Coma:	Blood 360-480 mg./100 c.c., urine 510-670 mg./100 c.c. Complete unconsciousness, depressed reflexes, subnormal temperature, anaesthesia, impairment of circulation, stertorous breathing, possible death.

This table has an "overlap" of 30 milligrammes at each end of the concentration ranges, but the main feature detracting from its value is the breadth of its concentration ranges. In Table II, by Purves Stewart (1937), this objection has been removed.

TABLE II.
Purves Stewart's Table.

0 mg. per 100 c.cm. (decil-litre) of blood	As in the normal abstainer. But also in many cases of <i>delirium tremens</i> .
80 mg. and downwards to 30, 20, 10 mg.	Show no gross clinical abnormality of behaviour. But towards the higher figures careful testing reveals delayed reaction time (0.4 second instead of 0.1 second) followed by hasty acceleration when driving a car, together with diminution of judgement, attention and control; also loss of efficiency in finer performance tests. The individual is more genial and has fewer inhibitions (" <i>In vino veritas</i> ").
100 mg.	33 per cent. show slighter pathological symptoms; talkativeness, euphoria, and abnormal confidence.
130 mg.	50 per cent. show similar clinical signs.
160 mg.	66 per cent. show similar clinical signs.
180 mg.	80 per cent. of cases show clinical signs of moderate intoxication, including diminution of control, mental confusion and disorders of coordination.
200 mg.	All cases (i.e., 100 per cent.) show marked inebriation with physical and speech disorders; also motor symptoms (cerebello-ponto-bulbar).
350 mg.	Stupor; inability to stand or walk.
400 to 500 mg. . . .	Coma ("dead drunk"), sometimes fatal.

Other tables of a similar nature have been published; but the difficulty is that the criteria of intoxication vary from worker to worker. A comparison of the figures of other workers, cited by Jetter (1938), places the blood alcohol concentration at which 100% of subjects show intoxication at a value near that of Purves Stewart (200 milligrammes per centum). The figures may be tabulated as follows:

Widmark, 592 cases: Intoxication in 100% of cases, 0.25 to 0.30 per centum (this is 250 to 300 milligrammes per 100 cubic centimetres of blood).

Schwarz, 905 cases: Intoxication in 100% of cases, 0.25 to 0.30 per centum.

Hoffman, 642 cases: Intoxication in 100% of cases, 0.20 to 0.25 per centum.

Harger *et alii*, 140 cases: Intoxication in 100% of cases, 0.20 to 0.25 per centum.

It will be noted that in the foregoing cases the clinical examination has been for intoxication, not for being "under the influence". Jetter, in an examination of 1159 hospital patients for acute alcoholic intoxication, found 83.6% of subjects who had a blood alcohol concentration of 0.20% to exhibit acute intoxication; his standard required, among other symptoms, that gross swaying, reeling or staggering should be present.

The time taken for attainment of maximum blood alcohol concentration will be seen from the work of different investigators to vary not only from person to person, but also in the one individual subject according to differing conditions. In the work of Miles (1923), the usual time taken to reach maximum blood alcohol concentration in his human subjects was from 75 to 120 minutes. When the same amount of alcohol was given in a solution which

was ten times the alcohol concentration, the time taken to reach the maximum was reduced in some cases to about 45 minutes, but in all cases a reduced time was recorded. It is not possible from the results of Miles to determine with any accuracy the time at which the maximum concentration occurred, because the points on the curve are too widely spaced at this region of maximum concentration; it is also noted that the amount of alcohol used in these experiments was small. One maximum concentration is recorded in the blood at 0.028%.

Most investigations have placed the maximum blood alcohol concentration in humans as occurring between 45 and 120 minutes after an initial dose of alcohol, the most favoured range being from one to two hours. After the maximum has been reached, the concentration of alcohol in the blood steadily diminishes provided no further alcohol is taken.

This behaviour is illustrated in Figure II. The blood alcohol curve is from an experiment conducted by myself, in which the subject consumed nine seven-ounce glasses of Victorian beer in twenty minutes. The maximum alcohol

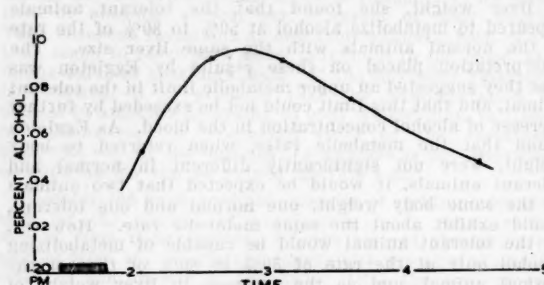


FIGURE II.

Graph showing blood alcohol curve of human subject. Maximum alcohol concentration 0.096% reached in sixty minutes.

concentration in the blood occurred in one hour after the cessation of drinking. It can be seen that for this initial dose just over 50% of the alcohol has disappeared from the blood in 160 minutes after the last drink. Higher concentrations will take longer periods to disappear from the blood. Newman, Lehman and Cutting (1937) show a curve in which the maximum concentration in a dog was 0.35%; approximately 80% of the alcohol had disappeared from the blood in eighteen hours.

Alcohol in Cerebro-Spinal Fluid.

Concerning alcohol in cerebro-spinal fluid, it has been held (Gettler and Tiber, 1927) that a true criterion of alcoholic intoxication is to be found in the alcohol concentration in the brain. Gettler and associates (1927, 1931, 1935, 1944, 1945) have pursued investigations from this aspect, and their works constitute some of the few which have involved cadaver examination. It has been stated by Gettler and Freireich (1931) that in dogs the alcohol content of the blood varies greatly when compared with that of the brain. According to these workers, the alcohol content of the spinal fluid is always somewhat higher than that of the brain, but there is always a definite and regular relation between the alcohol content of the two fluids. On the other hand, they assert that there is no regularity in the blood/brain ratios as there is in the spinal fluid/brain ratios. The analytical method used by Gettler and Freireich involved two distillations, and they found that "the optimum average recovery of alcohol from blood and spinal fluid to which known quantities of alcohol were added is 85 per cent". They used this factor of 100/85, based on 85% recovery of added alcohol, in their calculations of alcohol percentage in their experiments. Harger¹ gave the distribu-

¹Report to the National Safety Council, U.S.A., 1940; no longer available in Melbourne.

tion ratio of alcohol in cerebro-spinal fluid as 1:18 times that of blood, although later work (Harger, Hulpieu and Lamb, 1940) was not able to confirm a ratio as regular as this. The analysis of cerebro-spinal fluid has not been pursued as a means of determining alcohol concentration in the body.

Alcohol in Urine.

Alcohol is eliminated from the body in the urine, and there is widespread agreement that the amount so excreted is in the range of 1% to 4% of the total amount ingested. The reason for the interest in alcohol in urine has been from the point of view of determining whether the alcohol concentration in urine parallels that of the blood.

Miles, in 1923, compared the alcohol concentrations in venous blood from the arm and in the urine, and found that for the first two hours after the ingestion of alcohol there was no definite parallelism in the early stages of increasing alcohol concentration, but some forty minutes after this the urine concentration was from 40% to 50% higher. Smith and Stewart (1932) were unable to reconcile the amount of alcohol in the urine with the diagnosis of intoxication. They felt that insufficient attention had been given to the consideration that whereas a sample of blood at the moment of withdrawal shows an alcohol concentration representative of the time of withdrawal, a sample of urine "shows merely the average concentration of alcohol in the kidney secretion during a period of time". On the assumption—now shown to be incorrect—that the distribution ratio of alcohol between blood and urine was probably unity, they described three periods during which urine samples could be taken. First there is the period of increasing blood alcohol concentration, when the urine concentration will be less than that of the blood. Secondly, if the time during which the urine sample has been secreted covers the peak of alcohol absorption, the concentration of alcohol in the two fluids will be approximately equal. Thirdly, if the urine sample covers a period of diminishing alcohol concentration in the blood, the concentration of alcohol in the urine will exceed that of the blood. Smith and Purves Stewart concluded that "as a means of diagnosis of drunkenness urine analysis is valueless".

Haggard and Greenberg (1934) pointed out that if the alcohol in the blood passing through the kidneys came into equilibrium with the urine, the resulting concentration would depend on the relative solubility of alcohol in blood and urine. They also stated that the concentration of alcohol in urine bore no relationship to the volume of urine secreted, but that the amount of alcohol eliminated through the kidneys varied directly as the volume secreted. This means that of the total amount of alcohol ingested, the percentage lost through the kidneys depends upon the volume of urine passed. These workers investigated the point as to whether the kidneys exercised any selective secretory or absorptive action, and found that the concentrations of urine paralleled closely those of the arterial blood, but at a slightly higher level. This difference they found to correspond to differences of solubility of alcohol in blood and urine, and they fixed the ratio of distribution as 1:1.14 (blood:urine). A different light on the distribution ratio was supplied by Moritz and Jetter (1942), who observed that the urine/blood alcohol ratio varied inversely with the specific gravity of the urine (Table III):

TABLE III.

Specific Gravity of Urine.	Ratio, Blood : Urine.
1.020	1:1.30
1.030	1:1.23
1.040	1:1.16

Haggard, Greenberg, Carroll and Miller (1940) conducted two series of experiments to determine whether alcohol might be absorbed from the bladder in significant amounts under physiological conditions. They concluded that

absorption of alcohol from the bladder in man was of no practical importance at the concentrations in which it was likely to be found in ordinary testing. Moritz and Jetter, (1942) felt that the results of these experiments did not justify the assumption that the bladder was invariably an unaltered pool of ureteral urine. Of particular interest is their first experiment, in which ligation of the ureters of their experimental animals prevented urine from entering the bladder. Existing urine was withdrawn from the bladder and replaced with urine of lower alcohol concentration than the circulating blood. Analysis of blood and urine samples revealed that in each instance the concentration of alcohol in the blood fell continuously, whereas the concentration of alcohol in the urine rose. The greater the initial disproportion between the alcohol concentration of the blood and urine, the greater was the rate of increase in urinary alcohol concentration. As a result of a series of experiments it has been shown that during the period of rising alcohol concentration in the blood two processes may be contributing to the accumulation of alcohol in the urine of the bladder. One of these is the excretion of alcohol by the kidney, the other the diffusion of alcohol through the mucosa of the bladder. Once equilibrium has been attained, the latter will cease to be a factor.

It is evident from the foregoing that the alcohol concentration in the urine may be of value provided that the sample is taken after the blood has reached its maximum concentration. The present information on this subject indicates that a series of urine estimations may also provide information regarding the period of absorption of alcohol from the alimentary canal. If in two successive samples the concentration is falling in the urine, it may be inferred that the maximum concentration in the blood has been passed. If the two samples are of approximately the same concentration, or if the last taken is higher, then it may be inferred that these samples have been taken in that period when the concentration in the blood was at or near its maximum.

Summary.

The several decisions made by courts of law in Australia have not produced for the medical practitioner a clear definition of the condition of being "under the influence of intoxicating liquor". A Queensland court's description of the condition is that, as the result of consumption of liquor, the mental and physical faculties of a person are so affected as to be no longer in a normal condition.

The legal duties of a medical practitioner conducting an examination of a person suspected of driving "under the influence" are described.

Alcohol is absorbed unaltered from the stomach and small intestine and appears in the various body fluids. The maximum concentration of alcohol in the blood is reached within 45 to 120 minutes and it appears that to increase the alcohol concentration of the beverage consumed decreases the time taken to reach a maximum concentration in the blood.

After equilibrium has been reached, alcohol is distributed in the tissues so that the concentration is about 70% to 80% that of the plasma concentration. Fat contains a much lower concentration than other tissues.

Many workers have found that alcohol is eliminated from the blood-stream at a uniform rate, independent of the concentration. There is strong evidence, however, that at higher concentrations the removal of alcohol from the blood occurs at a greater rate.

A small percentage of ingested alcohol is eliminated from the body in the breath and urine. Oxidation in the body removes most of the alcohol.

The presence of food in the stomach has been reported to delay and reduce the maximum alcohol concentration in the blood. This effect is related to dietary protein; experiments with alanine suggest that a general rise in metabolism produces an increased rate of elimination of alcohol from the blood.

The position concerning tolerance to alcohol is not clear. It has been suggested that what may be taken as tolerance

is in fact a practised concealment of the effects of alcohol. Much work remains to be done in determining whether these are two separate factors.

References connecting blood alcohol concentration with degree of intoxication are described.

Alcohol concentration in urine is discussed. The force of the objections of earlier workers to the value of chemical tests with urine is diminished by the demonstration that diffusion of alcohol across the bladder mucosa may contribute to the urinary alcohol concentration.

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THE INFLUENCE OF NUTRITION ON THE GROWTH RATE IN THE FIRST YEAR OF LIFE OF CHINESE INFANTS BORN IN SINGAPORE IN 1951.

By JEAN MILLIS,

From the Department of Social Medicine and Public Health, University of Malaya, Singapore.

The gain in weight in Chinese infants in Singapore has been shown to be comparable with that of European children in other countries during the first six months. In the second six months the increase is slower, and at one year the Chinese infant is four to five pounds lighter (Millis, 1953 and 1954). This paper refers to information relating the feeding régime and external environment to the growth rate and physical condition of the child.

Collection of Data.

One hundred and twenty-eight infants were selected in the original study group, which was described in a previous publication (Millis, 1954). However, only 103 infants (57 males and 46 females) were under observation for twelve months, and it was decided to limit this discussion to the infants with complete records from birth to the end of the first year.

During each visit to the home for the routine measurement of the baby, the investigators questioned the mother regarding the feeding of the infant. Any alteration in the régime was noted, together with the date of the initiation of the change as accurately as the mother could remember. It was considered desirable to ascertain the effect of the prevailing practices of child care; therefore the investigators gave no advice or instruction in the food management of the infant. When the baby was weaned the reason for the cessation of breast feeding was recorded.

Each child was examined clinically at birth by the medical staff of the Kandang Kerbau Maternity Hospital, and at one year by Dr. Mary Grove-White of this department. Throughout the year the investigators noted all minor illnesses, such as colds, diarrhoea and skin eruptions, and this information was gathered by observation and by interrogation of the mother. If the illness was not considered to be of minor importance, the mother was advised to attend a Government welfare clinic or the general hospital.

Results and Discussion.

Infant Feeding.

Table I sets out the main categories of feeding encountered in the sample at different times during the year. At four weeks, 73 infants were entirely breast fed, 17 were artificially fed and the remainder were on a supplemented breast feeding. At twelve weeks the number of artificially fed infants had increased to 30 and only 50 infants were entirely breast fed. The desire to breast feed the infant is evident among these women, and 29 were still offering the breast when the child was one year old. The practice of prolonged breast feeding may be the result of the common belief that a woman will not conceive while she is feeding her child. Figure I compares the percentage of babies in this study who were fully and partly breast fed with the figures reported for Great Britain by Douglas (1950) and for Wuppertal by Dean (1951). Unfortunately Douglas gives no information of the ability of the women to feed their infants without supplements. The percentage of Chinese mothers who claimed that they provided some breast milk is high in the later months of the child's first year.

Thirty women had weaned their babies before the twelfth week, and the reasons for weaning are listed below: (i) failure of lactation, 21 cases; (ii) illness of the mother, six cases; (iii) the mother's absence from home at her work, two cases; (iv) indifference, which resulted in the adoption of the child by foster parents, one case.

These findings are in accord with those of Waller (1952), who states that the inability to establish a satisfactory milk supply is the chief cause of artificial feeding. In America, Darner and Hunter (1952) studied the effect of certain emotional factors on the initiation of breast feeding. They drew attention to the importance of rest and stated that physical and mental fatigue reduced the milk supply. The significance of this finding with reference to the Chinese women in the "sample" is unknown; but conditions in Singapore are certainly not conducive to rest or freedom from worry. The third-class wards in the maternity hospital are very overcrowded, and the babies are placed in

cribs at the foot of each bed. The mothers are discharged from hospital usually on the third to the fifth day after delivery, to return to cramped, noisy living quarters and the responsibilities and anxieties of their households. The hospital staff have little time to instruct the mother in the art of breast feeding, which is not properly established at the time of her discharge from hospital. It is perhaps surprising that the failures of lactation are so few.

It can be seen from Table I that a number of women found it necessary to supplement the supply of breast milk early in lactation. The diminished supply may have resulted from ignorance of the skills necessary for the

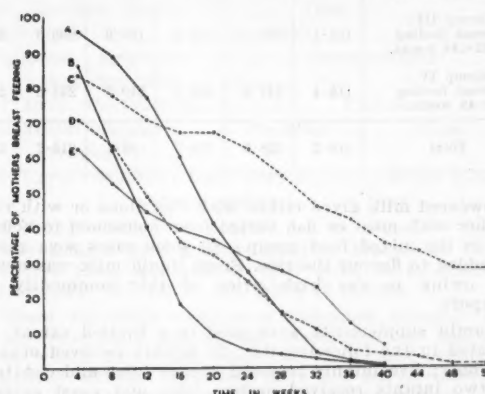


FIGURE I.

The percentage of mothers breast-feeding their infants in different countries: A, full and partial breast-feeding, Wuppertal; B, full breast-feeding, Wuppertal; C, full and partial breast-feeding, Singapore; D, full breast-feeding, Singapore; E, full and partial breast-feeding, Britain.

establishment of successful lactation or from under-nutrition of the mother. Kon and Mawson (1950) reported that the under-nourished woman in Britain lactated poorly, but the quantity rather than the quality of the milk was affected. Antonov (1947) found that the quantity of milk was reduced and that the length of lactation was shortened during a period of semi-starvation in Leningrad.

Sweetened condensed milk was preferred to powdered milk as a supplement to breast milk, but for artificial feeding little preference was shown. Only two mothers introduced starchy foods at sixteen weeks, but 31 babies were receiving rice, cornflour or oatmeal at twenty-four weeks, and of these foods a rice porridge called *Kanji* (Congee) was the most popular. After twenty-four weeks an increasing number of mothers offered meat or fish in the diet, but only two infants received vegetables at any time during the first year. A relatively large number of women continued breast feeding after the introduction of a mixed diet; but the contribution from breast milk could not be accurately assessed. The amount of sweetened condensed

TABLE I.
The Record of Feeding of Infants.

Feeding Régime.		Number of Infants in Age Groups (Four-Weekly Periods) with Distribution of Food Types.												
Breast Feeding.	Supplementary Food.	1	2	3	4	5	6	7	8	9	10	11	12	13
+	—	73	65	50	37	33	25	16	11	5	4	3	1	1
+	Sweetened condensed milk.	10	12	17	22	23	21	13	3	5	3	1	1	0
+	Powdered milk.	3	3	6	9	9	4	1	1	1	0	0	0	0
+	Starchy food.	0	0	0	1	3	7	10	8	7	7	7	4	3
+	Mixed foods.	0	0	0	0	1	7	16	24	25	22	23	23	25
—	Sweetened condensed milk.	9	11	16	17	14	7	8	7	4	6	5	4	5
—	Powdered milk.	8	12	14	16	14	16	9	6	5	4	1	1	0
—	Mixed foods.	0	0	0	1	6	16	30	43	51	57	63	69	71

TABLE II.
The Mean Weights in Ounces of Infants in Different Feeding Categories at Various Ages.

Feeding Category.	Age in Four-Weekly Periods.												
	0	1	2	3	4	5	6	7	8	9	10	11	12
Group I: Breast feeding <12 weeks.	110.6	131.3	150.7	182.9	204.3	219.6	231.9	244.3	252.3	263.0	271.7	278.8	288.8
Group II: Breast feeding >12 <32 weeks.	108.2	138.8	171.3	196.7	217.5	233.3	243.7	249.3	258.1	263.5	269.9	277.8	286.2
Group III: Breast feeding >32 <48 weeks.	104.1	135.2	164.3	187.9	206.3	222.4	233.3	244.2	250.3	257.1	262.9	270.0	271.7
Group IV: Breast feeding >48 weeks.	115.4	147.8	183.8	210.8	231.8	247.2	258.4	267.2	274.3	279.1	286.25	289.7	297.6
Total	110.2	138.5	170.2	195.1	215.7	231.3	240.1	252.0	259.6	266.6	273.8	280.1	287.6

or powdered milk given either with rice alone or with rice together with meat or fish varied from household to household in the mixed food group. In some cases soya sauce was added to flavour the rice. Fresh liquid milk was never used owing to the high price of this commodity in Singapore.

Vitamin supplements were used to a limited extent, as indicated in the following list: 28 infants received orange juice only; five infants received orange juice and cod-liver oil; two infants received orange juice and yeast extract ("Marmite"); one infant received cod-liver oil only; one infant received yeast extract ("Marmite") only. With regard to the 30 infants who were artificially fed before the twelfth week, eight received orange juice only, two orange juice and cod-liver oil, and two orange juice and "Marmite". Careful questioning of the mothers of the other 18 infants provided no information of any source of vitamin C in the diet at any time during the first year.

Judged by western standards, most of the infants were on a deficient diet after weaning. Of all the infants, 60% received no vitamin C in the first year other than that supplied by breast milk. The vitamin C content of breast milk is dependent on the level of vitamin C in the blood and therefore on the maternal intake. It has been shown that the vitamin C value of human milk undergoes seasonal variations in countries where there is a periodic shortage of fresh fruit and vegetables (Sinkko, 1937; Baumann, 1937-1938; Winikoff, 1946). Seasonal changes in supplies are not particularly noticeable in Singapore, but it is likely that the vitamin C level in the breast milk in this series was low. Many of the mothers observed the traditional taboos for thirty to forty days after delivery, which resulted in the exclusion of fruit and vegetables from the diet and caused a depletion of any reserves of vitamin C in the mother's body. Some women refused these foods for the time they were nursing, as they believed the consumption of fruit and vegetables would cause diarrhoea in the infant.

With the introduction of starchy foods nutrition begins to deteriorate, as the cereal is always highly refined, with a corresponding loss of nutrients. Eggs are usually avoided, as most of the mothers believe that an infant aged under one year will have a foul breath if eggs are consumed. Custom dictates that a child shall have two eggs on its first birthday; but this is by way of a celebration and not a lasting change in the dietary pattern. The quantity of meat or fish presented is very small, and therefore the intake of first-class protein falls when cereals replace milk as a source of calories. The calcium, vitamin A and riboflavin will also fall; but the intake of iron is likely to rise. At the age of one year, 32 infants on mixed feeding (31.1% of the sample) were receiving less than the equivalent of one pint of milk per day, and of these, six received less than half a pint. The infants had been on this low intake of milk for a relatively short time, and no obvious physical effects were observed at one year.

Weight Increase Related to Feeding.

It was decided to subdivide the infants into groups on the basis of the duration of breast feeding, and as the numbers are small the male and female infants will be considered together. The following classification was used. Group I: infants who were artificially fed before they were 12 weeks old (16 males and 14 females); Group II: infants who were transferred to artificial feeding after twelve weeks and before thirty-two weeks (14 males and 12 females); Group III: infants who were partly or fully breast fed at the thirty-second week and were weaned before the forty-eighth week (seven males and 11 females); Group IV: infants who were partly or fully breast fed for forty-eight weeks or longer (20 males and nine females).

The mean weight of each group at the different age levels was calculated, and this information is recorded in Table II. There was a significant difference in the birth weights of the infants in the different groups, therefore the results have been expressed as a percentage increase on birth

TABLE III.

Age.	Comparing Groups I and II.			Comparing Groups I and III.			Comparing Groups I and IV.		
	t	P	Remarks.	t	P	Remarks.	t	P	Remarks.
24th week	2.403	>0.01 <0.02	Significant.	2.215	>0.02 <0.05	Significant.	2.272	>0.02 <0.05	Significant.
28th week	1.425	>0.1 <0.2	Not significant.	2.017	>0.02 <0.05	Significant.	1.653	>0.05 <0.1	Doubtfully significant.
32nd week	1.564	>0.1 <0.2	Not significant.	1.792	>0.05 <0.1	Doubtfully significant.	1.518	>0.1 <0.2	Not significant.

weight in Table IV and Figure II. In all groups the babies had more than doubled their birth weights in the first six months. In this experiment, as in that of Dean (1951) at Wuppertal, for breast-fed infants there was no correlation between the gain in weight and the duration of breast

clinical examination was conducted by a university medical officer within a couple of weeks of the first birthday of the child. The medical officer was unaware at the time of the examination of the material collected about the child. The physical condition of the child was assessed by scoring on the following form:

- No. Name Sex Address
1. Skin: Dry Moist Infection, past, present
Cleanliness
 2. Subcutaneous tissues Firm Elastic
Dehydrated Abdomen
 3. Development: Walking Crawling
 4. Alertness: Bright Dull
 5. Colour: Pale Good
 6. Bones: Epiphyses Costo-chondral
Ant. fontanelle
 7. Eyes: Conjunctivæ Cornea
 8. Teeth: Number Condition Gums
Tongue
 9. Upper respiratory tract: Coryza Mouth breathing
Cervical glands Ears

Other comments

The infants were graded into four groups, as follows: (i) very good, four infants; (ii) good, 21 infants; (iii) satisfactory, 63 infants; (iv) poor, 15 infants.

There was little evidence of deficiency diseases in the group; one child had angular stomatitis, one had a dry skin which may have been due to a mild vitamin A deficiency, another child may have had active rickets, and one child was anæmic and had an enlarged liver. There was no clinical sign of scurvy; however, Pollis, Park and Jackson (1950), working on autopsy material at Johns Hopkins Hospital, found a number of infants aged under two years showing evidence of scurvy, although the disease had not been detected clinically. They were satisfied that in nearly all cases the scurvy antedated the terminal infection causing death.

Table VI compares the medical grading with the mean increase in weight in one year and the feeding category. The findings for weight increase agree with the general view that reasonable adequacy of body size and proportions and the rate of growth is related to physical health and fitness in children. It will be noticed that the duration of breast feeding has influenced the medical grading at one year. Although the distribution of infants in the grades "good" and "satisfactory" is approximately the same for all feeding categories, there were more infants from Groups III and IV classified as "very good" and fewer as "poor" than there were infants from Groups I and II.

Grulee, Sanford and Schwartz (1935), in a study of 2000 cases, showed that morbidity increased to the fifth month with all types of feeding and continued to rise to the ninth month among babies who were artificially fed from the

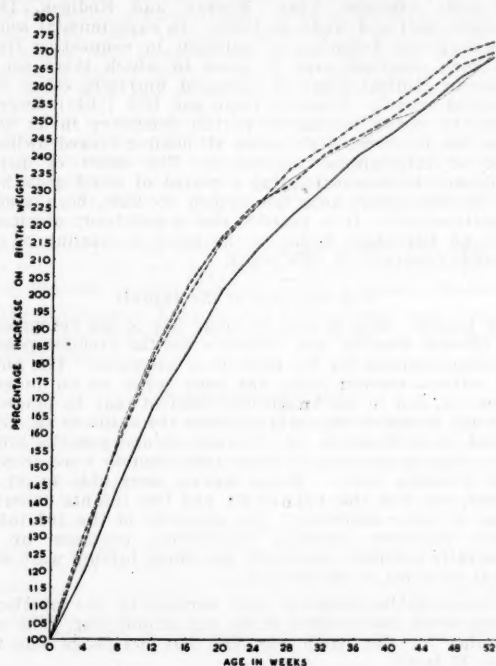


FIGURE II.

The percentage increase on birth weight of infants in different feeding categories: plain line, infants in Group I who were breast-fed for less than twelve weeks; interrupted line, infants in Group II who were breast-fed for more than twelve and less than thirty-two weeks; dots and dashes, infants in Group III who were breast-fed for more than thirty-two and less than forty-eight weeks; dots, infants in Group IV who were breast-fed for more than forty-eight weeks.

feeding. The average gain in weight of the breast-fed infants was significantly greater than that of the artificially fed infants at the twenty-fourth week, but not at the thirty-second week (Table III).

Medical History.

All the infants were classified by the medical staff of the hospital as full-term infants and normal at birth. A

TABLE IV.
Mean Weights Expressed as Percentage of Birth Weights of Infants in Different Feeding Categories at Various Ages.

Feeding Category.	Age in Four-Weekly Periods.												
	0	1	2	3	4	5	6	7	8	9	10	11	12
Group I: Breast feeding <12 weeks.	100	119.35	145.23	167.19	183.60	201.10	212.12	222.46	230.42	240.40	246.46	254.38	263.18
Group II: Breast feeding, >12<32 weeks.	100	128.77	159.12	182.99	202.36	216.36	226.49	231.70	240.08	245.08	251.19	258.48	266.23
Group III: Breast feeding >32<48 weeks.	100	130.46	158.99	181.87	199.59	215.86	225.63	236.46	242.52	249.31	254.92	261.78	264.50
Group IV: Breast feeding >48 weeks.	100	128.52	160.20	184.01	202.46	216.02	225.72	233.61	239.84	244.16	250.57	253.48	260.26

beginning. If the baby was totally or partly breast fed, the morbidity fell after the fifth month, and these babies appeared to be protected against gastro-intestinal disturbances.

In this study the data regarding the morbidity were collected by non-medical observers, and the less obvious deviations from the normal may not have been detected; also the conscientious mothers were more likely to report minor illnesses in the child in the time between visits. Only three infants had no record of illness during the year. Prior to the fifth month the incidence of minor illnesses was very small, but it increased rapidly in the sixth month and was maintained until the end of the year. The artificially fed infants, however, showed a greater susceptibility to all infections prior to the sixth month, and throughout the year the incidence was lower in the groups who were breast-fed longest (Table V).

TABLE V.
Incidence of Minor Illness.

Time in Four-Weekly Periods.	Number of Infants.			
	Group I (30): Breast Feeding 12 Weeks.	Group II (29): Breast Feeding 12 to 32 Weeks.	Group III (18): Breast Feeding 32 to 48 Weeks.	Group IV (29): Breast Feeding 48 Weeks.
1	1	1	0	1
2	2	1	1	0
3	3	1	0	0
4	2	1	1	0
5	9	4	1	3
6	13	10	7	12
7	11	14	5	5
8	12	15	7	11
9	11	13	7	9
10	11	11	4	6
11	14	9	7	13
12	9	10	6	13
13	15	18	7	11

Infections of the gastro-intestinal tract were at their highest incidence in the period from the twenty-first to the twenty-fourth week, with a subsequent gradual fall, although there was a small rise between the forty-fifth and fifty-second weeks. These infections corresponded approximately to changes in the dietary régime. Infections of the skin varied from prickly heat to boils of such severity that some infants remained infected for many weeks. The incidence was greater with increasing age after the sixteenth week. Infections of the respiratory tract became noticeable in the twenty-first to twenty-fourth week, and the incidence remained at approximately the same level until the end of the year. Eleven cases of measles were recorded, and in all cases except one the infant was artificially fed at the time of infection. Seven cases of ear infections were observed. It is reasonable to conclude that after the sixth month the progress of a number of the infants was retarded by infection.

In Singapore, vaccination against smallpox is compulsory before the child is six months old, and 72% of the sample were vaccinated between the twentieth and twenty-sixth weeks. It has been observed in the Government welfare

clinics that vaccination results in a temporary check in the weight increase of the infant.

It has been demonstrated in experimental animals that partial deprivation of vitamin C causes a delay in the healing of wounds in both the hard and soft tissues of the body (Bourne, 1946; Murray and Kodicek, 1946; Danielli, Bell and Kodicek, 1946). In experimental scurvy a widespread deficiency of collagen in connective tissue has been observed even in cases in which there are no apparent clinical signs of impaired nutrition other than retarded growth. Crandon, Lund and Dill (1940) observed adequate wound healing in partial deficiency in an adult man, but in severe deprivation all healing ceased owing to lack of intercellular substance. The effect of partial deficiency in humans during a period of rapid growth, as in the first year, does not appear to have been studied experimentally. It is possible that a deficiency of vitamin C is an important factor in the delay in healing of skin wounds observed in this study.

The Progress of the Infants.

A healthy child is able to make use of his resources in an efficient manner, and therefore has the greatest possible accomplishments for his level of development. The ability to perform certain tasks has been taken as an index of progress, and in the infant development may be judged by the age at which the child achieves the skills to sit, crawl, stand or walk alone. At the age of one year 25 infants were able to perform all these tasks and 68 were crawling and standing alone. Seven infants were able to sit and crawl, one was able only to sit, and two infants undertook none of these activities. The majority of the 103 infants were therefore making satisfactory progress by the generally accepted standards, and three infants were somewhat retarded in maturation.

Considerable variation was noticed in the number of teeth which had erupted at the age of one year. The mean number for the group was 5.94, but the range was from 0 to 20 teeth.

Housing and Hygiene.

Most of these families live in cubicles which are partitioned areas within a room. They are usually overcrowded and have inadequate light and ventilation, but nevertheless considerable effort is made to keep the cubicle clean and tidy. Cooking and washing facilities and sanitation are primitive, and are shared with other families in the same house. On western standards the level of hygiene is low. A detailed description of the housing is being prepared for publication. Variations in the standard of housing and hygiene within the group appeared to have no effect on the progress of the child.

Summary and Conclusions.

Fifty-seven male and forty-six female "full-term, normal" Chinese infants of the lower income groups were studied throughout the first year of life. They were divided into four groups on the basis of the duration of breast feeding.

The infants who were wholly or partly breast fed increased in weight more rapidly for the first twenty-four weeks than the artificially fed infants. In the breast-fed groups there was no correlation between gain in weight

TABLE VI.
The Medical Classification of Infants in Relation to the Method of Feeding.

Medical Classification.	Number of Infants.	Mean Increase in Weight in the First Year. (Ounces.)	Distribution of Infants in Each Feeding Category.			
			Group I.	Group II.	Group III.	Group IV.
Very good	4	209.6	1 (3.3%)	0	1 (5.6%)	2 (6.9%)
Good	21	190.7	6 (20.0%)	5 (19.2%)	3 (16.7%)	7 (24.1%)
Satisfactory	63	183.2	20 (66.7%)	14 (53.8%)	10 (55.6%)	19 (65.5%)
Poor	15	164.9	3 (10.0%)	7 (26.9%)	4 (22.2%)	1 (3.4%)
Total	103	183.1	30 (100.0%)	26 (99.9%)	18 (100.1%)	29 (99.9%)

and duration of breast feeding. There was no obvious benefit in gain in weight by prolongation of breast feeding after twenty-eight weeks.

The infants who were breast fed longer tended to be graded higher in the medical examination at one year. The incidence of minor illness prior to the fifth month was small, and such illnesses occurred more often in the artificially fed infants; but after the fifth month the morbidity rose rapidly. The high incidence of infection coincided with the slower rate of growth observed.

The diet of most of the infants was deficient in vitamin C, but no clinical signs of scurvy were detected. However, the lack of vitamin C may have contributed to the retardation of growth, the degree of lowered resistance to infection and the delay in healing of skin wounds observed in the second half of the first year.

At the end of the year 31% of the infants had less than one pint of milk per day and were on a diet low in first-class protein, calcium, vitamin C and probably riboflavin.

The standard of housing and hygiene was very low, and variations within the group had little effect on the progress of the child.

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FETAL AND NEONATAL ANOXIA IN RELATION TO OBSTETRICAL ANALGESIA AND ANÆSTHESIA.

By L. G. B. CUMPTON, M.B., B.S., M.F.A.R.A.C.S.,
Perth.

A CENTURY has passed since John Snow, using Sir James Young Simpson's technique of light chloroform analgesia, sat at the bedside of Queen Victoria allaying the pains of labour during the birth of her eighth child, Prince Leopold. The Queen's action in accepting analgesia was the determining factor responsible for overcoming the last vestiges of opposition by the churches, which up to that time said of women: "In sorrow thou shalt bring forth children." The Americans, however, claim that obstetric inhalation analgesia was first employed in 1847 by N. C. Keep, of the Harvard Dental School.

In 1880 nitrous oxide was used by Klikovich of Russia to relieve the pains of labour. Many other agents have had their vogue during the years—morphine, hyoscine, paraldehyde and ether, and more recently pethidine and trichlorethylene. In England, Minnitt's nitrous oxide and air machine has apparently given satisfaction for the past twenty years; yet only in recent years has Australia shown signs of adopting nitrous oxide as an obstetric analgesic.

One thus gathers that considerable attention has been paid to the mother. What of the infant? The United States Bureau of the Census reported the infant mortality rate during the first year of life as having declined from 100 per 1000 live births in 1915 to 40 per 1000 in 1942. It is further reported that neonatal mortality during the first week of life is 52% of the total mortality for the entire first year, and that during the first day is 29% of the total first year mortality. Australian figures are compared with these in the accompanying Tables I and II.

These figures show good progress, but that is not enough. It is my opinion that there is room for further improvement. More infants can be salvaged and the anaesthetist can and must help. With his knowledge of the pharmacology of analgesic drugs he can advise the obstetrician, he can spread practical hints among the nursing staff, he can cooperate with the paediatrician—in short, he must become an indispensable member of the obstetric team.

Factors Concerned in the Causation of Foetal and Neonatal Anoxia.

The Desire for Relief from Pain during Labour.

There is a desire, if not a demand, by most expectant mothers that they should be given relief from pain during labour. A natural corollary of this is the justifiable attempt by the obstetrician—occasionally with the help of the anaesthetist—to fulfil this want. Although the woman is anxious that her baby should be born healthy and well, she may during the throes of a stormy labour make all sorts of unreasonable demands for premature termination of it and also for relief of pain without thought of the possible harmful effects on the infant. Furthermore the medical attendant may be guilty of administering analgesic drugs in doses or at times likely to produce intranatal or neonatal anoxia. The point I am trying to make here is that, owing to the mere fact of the obstetrician's wanting to help the mother, he may irrevocably harm the child. With these facts in mind there would seem to be considerable merit in the Grantly Dick Read method of natural childbirth, which is said to be successful in about 50% of the patients selected as being suitable subjects for this technique. The other 50% do require some form of analgesia as well—either nitrous oxide or "Trilene"—especially at the transition from the first to the second stages of labour. Obstetricians will tell us that this method is gaining in popularity, and that there is an increasing demand by expectant mothers for natural childbirth. Are women by these demands casting, perhaps

¹ Read at a meeting of the Australian Society of Anaesthetists, Adelaide, April, 1953.

unconsciously, a slur on the ability of the medical profession? Or are they merely asking us to assist them in a psychological approach to pregnancy and labour? Or do they think we can relieve their pains by hypnosis?

Analgesic and Anaesthetic Agents.

Apgar and Papper (1952) state that all anaesthetic gases and vapours are known to cross the placental barrier—likewise morphine, pethidine and the barbiturates. There is no scientific proof but a very strong clinical impression that the relaxants do not traverse the placenta, or if they do, only in a negligible amount.

No obstetrician would consider giving morphine except in the early stages of labour, on account of its severe depressant effect on the foetal respiratory mechanism. Pethidine, on the other hand, is a far safer drug to use; not that it will not cause respiratory depression in the foetus—it does, but not to the same extent as morphine. Lee (1950) states that about 10% of infants require resuscitation, and that pethidine should not be given within three hours of delivery.

TABLE I.
Infant Mortality per 1000 Live Births: First Year of Life.

Year.	United States of America.	Australia.	Western Australia.
1915	100	67.5	66.5
1942	40	39.5	36.9
1949	—	25.3	26.4

Apgar and Papper (1952) quote Smith's determinations for nitrous oxide, cyclopropane and ether in maternal and foetal arterial and venous blood. Cyclopropane and ether traversed the placenta almost quantitatively, while only 60% of the nitrous oxide in maternal blood reached the foetus. There was a direct correlation of other levels with the depression of respiration. This conforms with our clinical impressions—the longer the anaesthetic administration especially with ether, the more sleepy and slow to inspire will the baby be (see Table IV). Cyclopropane is, of course, more rapidly eliminated than ether. Trichlorethylene rapidly enters the foetal circulation, but

TABLE II.
Infant Mortality, First Week of Life: Percentage of Total Mortality in First Year.¹

Year.	Australia.	Western Australia.
1949	61	64
1951	—	57

¹ The comparable United States of America figure is 52%.

has not been proved definitely to harm the foetus; probably this is because in the low concentrations used for analgesia it would merely stimulate the stretch receptors of the Hering-Breuer reflex—once respiration was established—and certainly would not depress the respiratory centre. Lee (1950) states that it is possible that drugs such as morphine or pethidine given to augment its action are at least partly responsible for the foetal respiratory depression.

Apgar and Papper mention the work of Hellman *et alii* in the transmission of thiopentone. According to their determinations in seven cases, before seven minutes and after twelve minutes minimal amounts were found in the blood of the new-born infant. Between these two intervals, the foetal level was 50% or more of the maternal level. This work was reported in 1944. It is now known that thiopentone is metabolized very slowly *in vivo* (8% to 15% per hour), and is stored in body fat. The state of hypnosis

(or subhypnosis) depends upon the plasma level at which equilibrium is established between fat and the rest of the body (Lief *et alii*, 1952). I would suggest that the apparent high level in the infant between seven and twelve minutes is probably only relative, as the maternal thiopentone has gone into fat storage by then. Lull and Hingson (1948) make the following statement:

From the standpoint of the pharmacologic effect upon the baby in utero, there is no method of pain relief in obstetrics which provides such safety through absence of toxicologic drug reaction and depression as spinal anaesthesia.

Be this as it may, subarachnoid and caudal block techniques have not found very great favour for routine analgesia in labour either in Australia or in Great Britain. One great disadvantage from the point of view of the baby is the increased incidence of forceps deliveries.

Spinal analgesia, however, has a place in obstetrics; according to Lee it is gaining in popularity as an analgesic method for Caesarean section. Adequate oxygenation of the mother—and hence the child—is essential. The blood

TABLE III.
Infant Mortality Under One Week for Western Australia, 1951. (Provisional.)

Causes of Death.	Number of Deaths.
Malformations	26
Injury (irrespective of maturity)	58
Asphyxia and atelectasis:	
Without mention of maturity	18
With immaturity	11
Pneumonia:	
Without mention of maturity	5
With immaturity	4
Associated with maternal toxæmia	16
Hæmolytic disease of new-born	11
Immaturity (unqualified)	89 (36.3%)
Other causes	7
Total	245

pressure will need to be controlled, especially if the analgesia reaches the costal margins, although the operation can be performed quite satisfactorily with analgesia to the level of the umbilicus. The role of spinal

TABLE IV.
Comparison of Nitrous Oxide and Oxygen with Other Anaesthetic Agents for Delivery, and the Effect on Respiration in the Infant (187 Cases).

Anaesthetic Agents.	Number of Cases.	Spontaneous Respiration in the Infant.
Nitrous oxide and oxygen	75 (40%)	90%
Ethyl chloride, ether, chloroform, cyclopropane	81 (55%)	67%

analgesia in the management of eclampsia has lately been emphasized by Lund (1952). Said to be due to spasm of the terminal arterioles, especially in the kidneys, eclampsia responds to conduction anaesthesia by a fall in blood pressure, cessation of convulsions, increase in urine output and a relief of headache. In 31 cases reported the maternal mortality rate was nil and the foetal mortality rate very low.

Physio-pathological States in the Mother.

There are two conditions, anoxia and hypotension, which should be treated as emergencies in the pregnant woman, as both may affect the oxygenation of the foetus. Of the several forms of anoxia, the anoxic type may occur during anaesthesia and the anæmic type may

seriously jeopardize fetal life in cases of *placenta prævia* and accidental hæmorrhage. The separation of the placenta provides a further hazard to the fetus in ante-partum hæmorrhage. In these cases, moreover, a general anæsthetic is usually needed, and this adds a further risk to the life of the fetus, which is often premature. Hence we have the triad of immaturity, anoxia and anæsthesia which, unless skilfully dealt with by prompt blood replacement, minimal anæsthesia and dexteros surgery, will result in a high fetal death rate.

As has been stated by Apgar and Papper, the effective placental blood pressure is the difference between the systemic arterial pressure and the intrauterine pressure. An appreciable fall or rise respectively may interfere with fetal oxygenation. Hypotension in the mother may result from spinal anæsthesia, and if it is severe it should be counteracted by means of a locally acting vasopressor, such as "Methedrine" or "Neosynephrine".

Fetal and Associated Conditions.

The fetus is said to be viable at the end of the seventh month, when it weighs about two pounds 12 ounces. At this stage of intrauterine life the lungs have developed sufficiently to take over the function of gaseous interchange of oxygen and carbon dioxide should the fetus be born. Even so the lungs are less mature than most other organs, owing to their slow development *in utero*. Although respiratory movements occur during intrauterine life, their function seems to be associated with the circulation of the amniotic fluid and certainly not with the oxygenation of the blood. Hence on premature birth we are faced with an underdeveloped organ—to wit, the lungs—upon which the very existence of the infant is dependent. It is not surprising therefore that these babies are extremely prone to *asphyxia neonatorum*. Furthermore, a depression of their respiratory centres by analgesic or anæsthetic agents can easily seal their fate. It is for this reason that conduction anæsthesia has been advocated for patients in premature labour (see Table III).

According to Potter (1952), the condition known as resorption atelectasis with hyaline membrane, which is often the cause of death in certain premature infants (1000 to 2500 grammes in weight) in the first twenty-four hours, particularly when they are delivered by Cæsarean section, is thought not to be caused by anæsthetic agents.

Such conditions as ante-partum hæmorrhage and cord entanglements are obstetric emergencies and may cause intrauterine anoxia of any degree according to the severity of the complication.

The most reliable clinical sign of fetal anoxia is progressive slowing of the fetal heart rate, and it should be treated by the immediate administration of oxygen to the mother pending any obstetric intervention.

Technical Ability.

As so many anæsthetics for obstetric procedures—particularly in private hospital practice—are administered by nurses under the supervision of the obstetrician, it is a wonder that more tragedies do not occur. Some of these nurses become quite good "practical anæsthetists" with both chloroform and ether after years of experience. Others are no doubt serving an apprenticeship. Should any anæsthetic emergency arise, the obstetrician would experience an anxious time trying to cope with a situation involving the lives of both mother and baby. To me it seems wrong that such a state of affairs should be accepted, tolerated and perpetuated. Unfortunately there is no complete solution to the problem, but there are several ways of improving the situation. Firstly, the obstetrician could administer the anæsthetic and leave the delivery to the nurse, who after all is trained to do such work. Secondly, the Grantly Dick Read method of natural childbirth will obviate the necessity for general anæsthesia at least in some cases. Thirdly, as inhalational analgesia with nitrous oxide and oxygen or "Trilene" is becoming more extensively used, and as nurses are being trained in

its administration, there is no reason why an increasing number of babies should not be delivered by this method.

Sequelæ of Anoxia.

Statistics (see Tables I and II) show a reduction over the years of the infant mortality rate. It is suggested however, that as anæsthetic agents and faulty anæsthetic administration must be regarded as factors contributing to neonatal anoxia and associated respiratory complications, some propaganda of an educational nature will aid in further reducing the neonatal death rate (see Table IV).¹

The effects of maternal anoxia on the infant's respiration may be set out as follows. In 187 cases there were six (3%) of maternal anoxia; of the infants, one was still-born and four were slow to inspire.

The number of post-natal complications following the use of nitrous oxide and oxygen and other anæsthetic agents was as follows. There were in this series 169 cases; post-natal complications followed nitrous oxide and oxygen anæsthesia in one case, and other forms of anæsthesia in seven cases (including one death).

We should consider not only the mortality rate or immediate effects of anoxia, but also the all-important matter of morbidity or remote results. Whereas the immediate effects are mostly clear cut, the late effects of anoxia may have to be regarded as more a matter of probability and surmise. There is, however, some suggestive clinical evidence according to Little, Hampton and White (1952) that intranatal and neonatal anoxia causes cell damage in the cerebral cortex so that the subject's mental ability is accordingly reduced. If this is so, one may expect every grade of cortical destruction from very slight to very gross, dependent on the degree and period of anoxia, and this may provide one important factor in accounting for the various levels of mental intelligence. (Perhaps those who can boast a high intelligence quotient should be thankful to the obstetrician as to their forebears.)

Preventive Measures.

Sufficient has been said to stress the significance of the relation of anoxia in the newborn to anæsthesia and analgesia. There are now several points of a prophylactic nature which need emphasis.

Firstly, according to Lee (1950), the use of nitrous oxide and air over a long period of time cannot be pronounced entirely guiltless in the matter of fetal hypoxia. This provides only 10% oxygen when a 50-50 mixture is used. A much more desirable mixture would be one in which the oxygen percentage was not less than the atmospheric percentage. The nitrous oxide and oxygen machines in current use in at least two Australian States are fitted with a locking device that permits the delivery of not less than 30% oxygen. The C.I.G. Type A intermittent flow machines installed at the King Edward Memorial Hospital (Subiaco, Western Australia) have this safety device and are similar to those in use at the Women's Hospital, Melbourne, with the exception that ours are fixed to the wall at the head of the bed; this minimizes and facilitates cleaning of the labour ward cubicles.

Secondly, in cases of fetal distress, evidenced by a progressive slowing of the fetal heart rate, the immediate administration of oxygen to the mother is a simple emergency measure that can be carried out by any nurse, pending the adoption of the necessary obstetric treatment.

Thirdly, the introduction of a new drug, N-allylnormorphine, as an antagonist to the respiratory depression caused by opiates and pethidine, may prove invaluable in preventing neonatal anoxia. It has also proved effective in the infant when injected into the umbilical vein. This investigation was carried out by Eckenhoff, Hoffman and Dripps (1952). Shaw and Bentley of Melbourne (1949)

¹ The figures in Table IV, and those relating to the effects of maternal anoxia on the infant's respiration and to post-natal complications following different types of anæsthesia, are taken from a small series of cases at the King Edward Maternity Hospital.

have shown that "Monacrin" is an excellent antidote to the respiratory depression of morphine in dogs.

Fourthly, the caudal and spinal techniques as developed and practised by Lull and Hingson of the United States have much to commend them as having minimal depressant effects on the fetus, and perhaps should be more often employed in selected cases.

Fifthly, in the use of general anaesthesia for Caesarean section one feels that speed in extraction of the infant is to its advantage. This would ensure a minimum of drugs reaching the fetus before its separation, provided that the anaesthetist withheld induction of anaesthesia until the drapes were being applied. The intravenous administration of thiopentone and a relaxant with cyclopropane maintenance is the technique I employ. This enables the surgeon to make the incision within about a minute of the induction, and usually to extract a spontaneously crying infant in five to ten minutes. The practice of pre-operative rupture of the membranes, said to minimize the aspiration of liquor amnii by the fetus, should be of value in reducing the incidence of asphyxia neonatorum. However, I cannot speak with any experience of such cases.

Conclusion.

In conclusion, I should like to state that my object in presenting this paper is to stimulate interest and thought in this branch of anaesthesia, with the aim of helping to prevent the not inconsiderable wastage of young lives, and to reduce the cerebral morbidity rate which, as I have suggested, may be due to foetal and neonatal anoxia.

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Reports of Cases.

PSYCHOTIC MANIFESTATION OF CORTISONE INTOXICATION: A CASE REPORT.

By F. G. RAINSFORD, M.B., B.S.,
 Resident Medical Officer, Sydney Hospital.

ALTHOUGH recent literature has contained many hundreds of articles on various aspects of cortisone, comparatively few stress the very real possibility of toxic effects from this drug.

Rome and Braceland reported a series of cases in which a prepsychotic personality gave rise to a frank psychosis on the administration of cortisone. Tournay and Gottlieb described four patients in whom the psychotic effects were considered due to "the drug, premorbid personality trends, and psychological meaning of therapeutic failure". Clark *et alii* recorded a series of four minor and six major cases of mental disturbance occurring during ACTH and cortisone therapy.

This report is of interest, in that the mental symptoms appeared only on the cessation of therapy and appeared to be a true withdrawal effect.

Clinical History.

Mrs. X, aged fifty-eight years, was admitted to Sydney Hospital with a history of having received a total of 3.1 grammes of cortisone during the previous seven weeks for an arthritic condition of the hips. Three days before her admission to hospital the patient had developed some generalized oedema, whereupon cortisone administration had been immediately stopped. The oedema subsided over the next twenty-four hours after the injection of 1.5 cubic centimetres of mersalyl. Thirty-six hours after she had received the last dose of cortisone, the patient's family noticed that she had become confused in thought and speech. During the next twelve hours there developed persecutory delusions directed towards her husband and other members of the family.

On examination on her admission to hospital, the patient was non-cooperative, confused and irrational. No other abnormalities were detected in the central nervous system, although the eliciting of the deep reflexes in all limbs appeared to produce pain and she cried out. These reflexes were quite normal. There were minimal signs of dehydration. Examination of other systems revealed no abnormalities.

During the next four weeks the patient remained confused and disorientated in time, and suffered from continual auditory hallucinations, usually in the form of the hearing of voices of close relatives. She was especially abusive towards her husband on the occasions when she recognized him. Prior to her illness she had spent many happy years with him. At the end of four weeks the patient revealed one morning that she realized she was in Sydney Hospital. From that point onwards her mental condition improved rapidly, and one week later she was considered fit for discharge.

During her stay in hospital the following tests were carried out, and in each case the results were reported as being normal or within normal limits: (i) serum sodium, potassium and chloride estimations; (ii) hemoglobin estimation and blood films and counts (iii) estimation of the carbon-dioxide combining power and of the blood sugar content; (iv) cerebro-spinal fluid examination; (v) daily blood-pressure readings and urine testing; (vi) an electroencephalographic examination; (vii) microscopic examination of the urine; (viii) a Wassermann test. The albumin-globulin ratio on her admission to hospital was 2.9:2.3. After one week this ratio had returned to normal, and it remained so for the rest of the period in hospital. The electroencephalogram revealed a generalized disorganization of the normal cortical rhythm from all areas, but there was no indication of localized abnormal discharge.

Treatment was symptomatic. Hydration was carefully watched and nourishment was provided through a Ryle's tube when indicated.

A two-months' follow-up after the patient had left hospital indicated that she was in good spirits, with no subsequent residue of her psychosis.

Comment.

It was suggested that if this was a true withdrawal effect the patient should be treated with small doses of cortisone. However, in the interests of the patient, this treatment was not considered to be safe or warranted.

A further suggestion for using electro-convulsive therapy was not carried out, as it was thought that the patient was making slow but definite progress with conservative treatment.

Acknowledgement.

I wish to thank Dr. K. B. Noad for his permission to present this case, and for his encouragement.

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ELECTRICAL DEFIBRILLATION OF THE HEART.

By R. H. ORTON, M.B., B.S., F.F.A.R.C.S., F.F.A.R.A.C.S.,
D.A.,

*Director of Anaesthesia and Resuscitation,
Alfred Hospital, Prahran,
Victoria.*

NUMEROUS reports have appeared in overseas medical journals of equipment designed for the purpose of defibrillating the heart when ventricular fibrillation occurs. However, to the best of the writer's knowledge, no successful case has been recorded in the Australian literature. It was considered advisable, therefore, to publish the following case report.

Clinical Record.

E.E., a female patient, aged fifty-nine years, was admitted to the Alfred Hospital for the operation of mitral valvotomy. An electrocardiographic tracing showed that the heart was in the state of auricular fibrillation, at an average rate of 72 beats per minute. The average blood pressure was 135 millimetres of mercury, systolic, and 90 millimetres, diastolic.

Pre-operative testing of the patient with sedatives revealed extreme sensitivity to morphine and its derivatives. Accordingly pre-anæsthetic sedation consisted of "Seconal", 0.1 gramme, given one hour before operation. An intravenous infusion of blood was started and anaesthesia was induced, with the patient in the sitting position, by the intravenous injection of 0.15 gramme of thiopentone. When the patient was asleep, oxygen was administered and the patient was lowered into the recumbent position. An intravenous drip administration of "Brevitol M" was commenced at a rate of 12 milligrammes per minute. After two minutes laryngeal intubation was performed and anaesthesia continued with nitrous oxide (80%) and oxygen (20%), controlled respiration being used. The infusion of "Brevitol M" was slowed to approximately four milligrammes per minute, at which rate of flow the laryngeal reflex was just abolished. The operation proceeded normally until the surgeon's finger was passed through the mitral valve. It was then noticed, both by direct observation of the heart and by the electrocardiograph, that ventricular fibrillation had occurred. The surgeon was asked to withdraw his finger into the auricle, but it was found that the ventricular fibrillation continued. Accordingly the finger was withdrawn from the heart, a clamp was applied to the auricle and cardiac massage was begun. After about two minutes, during which time the patient's lungs were rhythmically inflated with 100% oxygen by the anaesthetist, it was decided that defibrillation of the heart was essential. Accordingly the electrodes of the defibrillator were applied across the heart, and two shocks each lasting 0.05 second were given in quick succession. The surgeon then noted that the heart was in standstill, but a third shock was administered before the anaesthetist could be notified. Immediately after the third shock the heart commenced to beat with a normal rhythm and the electrocardiographic tracing resembled that taken before operation, except for some depression of the S-T segment. The rate of the heart was slow at first, but this was soon followed by a short period of tachycardia. The electrocardiograph was watched for several minutes, and when the tracing returned to normal the heart was sutured and the operation completed. The patient regained consciousness immediately after removal of the endotracheal catheter, and apart from a feeling of general weakness for two days, showed no departure from normality. There was no evidence of cerebral damage.

Comment.

The defibrillator used was constructed in the Baker Research Institute of the Alfred Hospital, and its efficiency had previously been demonstrated by animal experiment. The instrument was designed according to the principles described by Mackay, Mooslin and Leeds (1951), with certain modifications. This instrument provides a shock of 110 volts alternating current, with complete isolation of the patient from the electrical mains by the use of two transformers. Timing is automatic and is made by a "Thyatron" valve with variable resistance-capacitance networks. The shortest impulse lasts 0.05 second and the longest 0.5 second. Repeated shocks are given by switching from "stand-by" to "shock".

Reference.

- MACKAY, R. S., MOOSLIN, K. E., and LEEDS, S. E. (1951), "The Effects of Electric Currents on the Canine Heart, with Particular Reference to Ventricular Fibrillation", *Ann. Surg.*, 134: 173.

Reviews.

Pathology for Students of Dentistry. By George L. Montgomery, T.D., M.D., Ph.D., F.R.F.P.S. (G.), F.R.S.E.; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 305, with 133 illustrations. Price: 37s. 6d.

In his preface Professor Montgomery states that there are many difficulties in presenting a key subject within a restricted time to students otherwise engaged in a busy curriculum. The balance of the whole course must be considered, and the presentation must be concise but not superficial. He has made an admirable attempt at producing a course of instruction for dental students which is not just a series of excerpts from the medical course. He has tried, where possible, to illustrate general pathological changes by reference to the mouth, which has been a field where technical accomplishments have overshadowed the recognition of biological principles.

Some aspects of pathology which are irrelevant to the dental course have been omitted; but on the other hand the attempt to be concise may lead to one or two misconceptions. For example, it can hardly be said that "suppuration is the formation of an abscess in the tissues". Some chapters might be expanded. The discussion of the anemias and leuchemias has been unduly curtailed, and there is scant reference to Hodgkin's disease. Disorders in calcium and phosphorus metabolism and hypertension are subjects which could well be treated, instead of the devotion of a chapter to peptic ulceration.

No claim is made to discuss the special pathology of the mouth; but the author does well to give a clear outline of the known facts of periodontal disease without reference to the mass of hypotheses under which they are hidden in dental literature. It is to be regretted that a statement in similar terms is not made on the subject of dental caries. An early appreciation of the histopathology of this disease would greatly assist the student in his later dealings with it.

This book is well written, and apart from these few criticisms, can be highly recommended to dental students.

Diseases of Women. By Ten Teachers under the direction of Frederick W. Roques, M.D., M.Chir., F.R.C.S., F.R.C.O.G., edited by Frederick W. Roques, John Beattie and Joseph Wrigley; Ninth Edition; 1953. London: Edward Arnold and Company. 8½" x 6", pp. 488, with 177 illustrations. Price: 28s.

This book is well set up, adequately illustrated, and printed in clear type on excellent quality paper. When ten teachers of such calibre as these authors produce a textbook, their combined effort must of necessity be authoritative in the medical world. In this ninth edition improvement is noted, in that each author is allowed more individual scope, so that teaching dogmatism is made much easier. The first chapters are devoted to the anatomy and physiology of the female pelvic organs, and it is interesting to note that a complete chapter is utilized to describe the anatomy of the pelvic floor, its importance being thus stressed. There is a good summary of the sex hormones and their uses (without undue claims for success), and this has been brought right up to date to include such therapy as the use of implants.

From a practical teaching viewpoint the book is to be commended for its division into chapters, each discussing a clinical entity or symptom.

An unusual but very valuable chapter is devoted to neurasthenia and neurosis in relation to pelvic disorders. If this can be absorbed early in a doctor's career, it should prove of inestimable value to him in practice.

The last few pages of this book are given over to operative gynecology, as if it were an afterthought. The operations are so sketchily described that it might have been better to omit this section altogether and refer the reader to another text-book on this subject.

There are, of course, many minor points with which we could register disagreement. For instance, insufflation of the Fallopian tubes is considered safe only in the operating theatre and under anaesthesia, whereas this must surely be an exceptionally elaborate method of procedure.

Perhaps the authors have on purpose kept to a somewhat extreme simplicity throughout the book; but this rather restricts it to students and young practitioners. To them, however, it can be confidently recommended as a lucid, straightforward and complete summary of present-day sound gynecological teaching.

Notes on Books, Current Journals and New Appliances.

Family Doctor. Published monthly by the proprietors, the British Medical Association, Tavistock Square, London, E.C.1. Sole agents for Australia and New Zealand: Gordon and Gotch (Australia), Limited. Subscription for twelve months: 20s. (sterling), including postage.

A CHUBBY rosy infant, symbolic of the new year, gazes into the future from the front cover of the January *Family Doctor*. The contents are well up to standard, articles ranging in their subject matter from hypnotism to invalid cookery. Walter Alvarez contributes the second of his common-sense articles on "How to Live with your Nerves". J. Gordon Cook continues his interesting series on popular science. Glimpses are given of children on the stage and their welfare, of old folk obtaining congenial employment and of the Emergency Hospital Bed Service in London. Various medical and psychological problems are dealt with in a helpful fashion. This is all put together in the usual attractive way to make a magazine that any doctor can recommend to his patients with confidence. A free sample copy of *Family Doctor* will be sent to any medical practitioner on request to the Editor of THE MEDICAL JOURNAL OF AUSTRALIA.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Ten Lepers from Naestved in Denmark: A Study of Skeletons from a Medieval Danish Leper Hospital", by Vilhelm Møller-Christensen, M.D., with a preface by Erik Waaler; 1953. Copenhagen: Danish Science Press, Limited. 10½" x 7", pp. 162, with 119 illustrations. Price: \$5.00.

The skeletons studied were from 400 to 700 years old.

"The General Practitioner's Guide to Physiotherapy", by Janet Dennison, M.C.S.P.; 1953. London: William Heinemann (Medical Books), Limited. 7½" x 5", pp. 46. Price: 6s.

Consists of short chapters on massage, exercises, electrotherapy and manipulation with a list of ailments with the type of physical therapy which may be used in each.

"Michael Servetus: A Translation of his Geographical, Medical and Astrological Writings with Introductions and Notes", by Charles Donald O'Malley; 1953. Philadelphia: American Philosophical Society. London: Lloyd-Luke (Medical Books), Limited. 9" x 6", pp. 208, with seven illustrations. Price: 21s.

The aim has been to produce a literal translation rather than an elegant rendering.

"Pediatric Gynecology: With Sections on Urology and Proctology", by Goodrich C. Schauffler, M.D.; Third Edition; 1953. Chicago: The Year Book Publishers, Incorporated. 9" x 6½", pp. 318, with 76 illustrations. Price: \$7.50.

The book "is calculated to be clinically helpful more than scientifically exhaustive".

"The Year Book of Obstetrics and Gynecology (1953-1954 Year Book Series)", edited by J. P. Greenhill, B.S., M.D., F.A.C.S.; 1953. Chicago: The Year Book Publishers, Incorporated. 8" x 5½", pp. 568, with 104 illustrations. Price: \$6.00.

One of the "Practical Medicine Series" of Year Books.

"Anatomy and Surgery of Hernia", by Leo M. Zimmerman, M.D., and Barry J. Anson, Ph.D. (Med. Sc.); 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10½" x 7", pp. 384, with many illustrations. Price: £5 7s. 6d. from The Williams and Wilkins Company.

The anatomical studies of this book are the result of questions raised by surgeons.

"Clinical Disorders of the Heart Beat", by Samuel Bellet, M.D.; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson. 10½" x 7", pp. 376, with 164 illustrations. Price: £4 11s. 6d. from Lea and Febiger.

Deals with "the pathophysiology, clinical and electrocardiographic findings and recent measures employed in therapy" of disorders of the heart beat.

"Gynecological and Obstetrical Anatomy and Functional Histology", by C. F. V. Smout and F. Jacoby; Third Edition; 1953. London: Edward Arnold and Company. 8½" x 5½", pp. 344, with many illustrations. Price: 35s.

The first edition was entitled "The Anatomy of the Female Pelvis"; the second was "Gynecological and Obstetrical Anatomy". The present title indicates the inclusion of "the dynamic histology of the female reproductive system and its endocrine control". A synopsis of progress in midwifery has been added.

"A Text-Book of Pathology: An Introduction to Medicine", by William Boyd, M.D., Dipl. Psych., M.R.C.P. (Edin.), F.R.C.P. (Lond.), F.R.C.S. (C), LL.D. (Sask.), D.Sc. (Man.), M.D. (Oslo), F.R.S. (C); Sixth Edition; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" x 6", pp. 1024, with many illustrations, some in colour. Price: £6 14s. 6d. from Lea and Febiger.

The first edition was published in 1932. Since then there have been a total of 21 reprintings. The book has been translated into Spanish and Portuguese.

"The Autonomic Nervous System", by Albert Kuntz, Ph.D., M.D.; Fourth Edition; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 8½" x 8", pp. 606, with 34 illustrations. Price: £5 7s. 6d. from Lea and Febiger.

The author tries to cover the anatomy, physiology, pathology and clinical relationships of the autonomic nervous system.

"Common Diseases of the Ear, Nose and Throat", by Philip Reading, M.S. (Lond.), F.R.C.S. (Eng.); Second Edition; 1953. London: J. and A. Churchill, Limited. 8" x 5½", pp. 296, with 39 illustrations, two in colour. Price: 22s. 6d.

The first edition of this book was reviewed in these pages in April, 1951.

"Fearless Childbirth: What Every Mother-To-Be Should Know", by Minnie Randell, O.B.E., S.R.N., S.C.M., F.C.S.P. (Hon.), with original illustrations by Dorothea Farewell; Second Edition; 1953. London: J. and A. Churchill, Limited. 7½" x 5", pp. 112, with 51 illustrations. Price: 3s. 6d.

A second edition of the booklet reviewed in these pages in March, 1949. Several minor additions have been made.

"The Quest for Youth: A Study of All Available Methods of Rejuvenation and of Retaining Physical and Mental Vigour in Old Age", by George Ryley Scott, F.Ph.S. (Eng.), F.Z.S., F.R.A.I.; 1953. London: Torchstream Books. 8½" x 5½", pp. 158. Price: 10s. 6d.

"The secret of living is to enjoy all the years that make up one's existence, to remain physically and mentally active until the end comes."

The Medical Journal of Australia

SATURDAY, FEBRUARY 27, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE BRITISH COUNCIL.

In November, 1947, attention was drawn in these columns to the work of the British Council, mainly because people seemed to have little idea of what the Council stood for and what its real purpose was. It was pointed out that the Council was inaugurated in 1935 by the Government of Great Britain, with funds provided by parliamentary grants for the purpose "of making the life and thought of Britain more widely known abroad". Stress was laid on the fact that there was no intention to project the British way of life, but that the work of the Council was to be only one side of an exchange. Because it was not desired that the Council should in any sense be an instrument for propaganda of the government of the day, its affairs were placed in the hands of an executive committee which was representative of all branches of current thought. Ultimately it was decided that the Council's scope would be restricted to educational and cultural work; this was done in order to avoid overlapping between the Council and the Government's overseas information services. Perhaps readers do not need to be reminded that one of the main activities of the Council from the medical aspect is the production of the *British Medical Bulletin*, a journal which records work done in Great Britain in the medical sciences.

The Council's report for the year ended March 31, 1953, has been published. It is an interesting and revealing document. Perhaps the most important point about this report is that the Council has been compelled to carry on its activities on a reduced expenditure. The grant for the year under review was £2,527,100—a decrease of £223,000 on the amount allowed for 1951-1952, an 8.1% reduction. The President of the British Council is Sir Henry Dale, O.M., and the Executive Committee numbers some 22 persons who are representative of many different cultural activities. To give an idea of the range of activities covered

by the Council it may be pointed out that the following panels and committees exist. The panels are: "Books and Publishing", "Editorial Advisory", "English Studies", a Scottish panel and a Welsh panel. The committees are those of "Drama", "Fine Arts", "Law", "Music", "Science", and "Universities". The science committee comprises committees dealing with agriculture, medicine, science and engineering, and veterinary science. The overseas representatives living in different countries number between 50 and 60. Australia has a representative whose office is in Sydney. The number of overseas persons who have visited Great Britain for purposes of professional study and with whose visits the British Council was associated was 3500 for the period under review. The number coming from the British Commonwealth was 670. The number from the Colonies was 306, from the United States of America 257, from Latin America 306, from the Middle East 406, from the Far East 221, from East and Central Europe 337, from North and Western Europe 766, and from South Europe 350. Of these visitors, 481 came to study medicine, 770 were concerned with education, 591 with science, and no less than 1097 with social science. During the period 1947-1948 over three-quarters of the visitors dealt with by the Council were its guests, and the total cost to the Council was £67,000; in 1952-1953 less than one-sixth of the visitors received financial assistance, the cost falling to £8000. Although the Council has not been able to expend more of its own funds in this way, it has administered in the United Kingdom the fellowship and scholarship programme of the United Nations and its specialized agencies, it has placed foreign government and private scholars, and it has run self-financed courses and study tours, and has made programme arrangements for individual professional visitors who had come to Great Britain at their own expense. What is described as the most interesting feature of the period in this regard was the rapid growth of the United Nations fellowship and scholarship scheme. In 1947-1948 there were 33 awards of this kind in the United Kingdom. In the year under review they had risen to 362. It is pointed out that at the time the report was written there were 18,000 overseas students in Great Britain. These students would spend something like £7,000,000 a year in fees and maintenance while they followed their training in Great Britain. It is understandable that some emphasis is placed on this economic aspect of the business. The report goes on to say that apart from any moral duty a highly developed country such as Britain may have to place its training facilities at the disposal of the world, and it is a matter of enlightened self-interest that it should do so. "Nowhere is this seen more clearly than in the Commonwealth and Colonies." Then it is added that as imperial ties weaken, strong cultural links are being forged.

In a chapter headed "Work in the Commonwealth" the work in Australia is set out in one short paragraph. It is stated that books for children selected by the Council and by "Books of Our Time" have been circulated by the Australian Library Association. In Sydney and Melbourne the Council arranged the exhibition of the development and use of *The Times* new Roman type face sponsored by the Printing Industry Craftsmen's Association of Australia. The Council also arranged a six months' tour by Michael Langham in Western Australia, adjudicating at the

Western Australia Theatre Council Drama Festival, and advising on production, all expense in Australia being paid by the Adult Education Board of Western Australia. A similar tour by Robert Speaight was arranged in collaboration with the Australian Broadcasting Commission.

By far the most important chapter in this report is that describing work in Europe. Unfortunately the Council's work in Europe during the year under review reached its lowest financial level since the war. This was due not only to closure of provincial centres and further reduction of staffs in a number of countries, but to the general reduction of supplies and of personal exchanges which everywhere followed the reduction of the Council's grant-in-aid. Only in Germany and Yugoslavia was the level of activity maintained or even slightly increased. We read that even such a fundamental need as the replenishment of the Council's libraries abroad is not being met and, more important still, the European demand for cultural contact with Britain is very far from being satisfied. This European demand is described as the most active element in the situation, and the suggestion is that it is worth considering whether even from a narrowly national point of view a more sociable attitude towards cultural cooperation might not help to "ease some of our political and economic differences with our European friends". It is stated that it would be naïve to suppose that an increase in the number of lectures, orchestras or works of art sent abroad would entirely remove the disappointment, however unreasonable, which British policies at some times caused. On the other hand, it would be shortsighted not to realize that a friendly response to an invitation to strengthen cultural links with Europe would help to take the sting out of refusals in other directions. This is not simply a superficial question of method or of manners; it is claimed to touch three points at least on which European opinion is extremely sensitive. First, there is the general importance attaching to organized cultural activity in European eyes; secondly, there is a specific importance attached to cultural exchange in the various plans for the rehabilitation and the spiritual defence of Europe; finally there is the question of obligation under the cultural conventions signed with the various European powers. Many Australian readers will find it hard to understand the fact that in Germany opera houses and museums were often rebuilt after the war before homes for the people. It is not to be wondered at that British observers in Germany "were astonished and even shocked" at this. The post-war multiplication of festivals all over the Continent is not merely a device for encouraging tourist traffic, although this is stated to be one of the objectives. "It is one of the symptoms of the desire, partly conscious, partly instinctive, to restore the cultural circulation of Europe—one might almost say, the cultural circulation which is Europe." What have been termed cultural conventions have been brought into existence, and of these there are nine. The general purpose of a cultural convention is described as that of "promoting by friendly interchange and cooperation the fullest possible knowledge and understanding in their respective countries of the intellectual, artistic and scientific activities, as well as of the ways of life of the other countries". The following nine forms of cooperation are set out: the promotion of the study of each other's language, literature and history in universities and other

educational institutions; the free establishment of cultural institutes in each other's territory; the interchange of university teachers, school teachers, students and school pupils; the provision of scholarships and bursaries; cooperation between learned societies and educational and professional organizations, including technical and social organizations such as trades unions; an attempt to equate as far as possible the academic degrees and awards and the standard professional qualifications required in one country with those of the others; the development of holiday courses; the encouragement, by invitation or subsidy, of reciprocal visits by cultural, technical and professional groups; and the joint propagation of a knowledge of each culture through the medium of the arts and of the printed and spoken word.

The report states quite frankly that Britain realizes that she has much to give, probably more than other countries, in the way of cultural and educational accomplishments. Each country gradually acquires or builds up its own form of culture, but no one would deny that the building is based on ideas received from other sources, particularly the sources from which the country concerned has originally sprung. It is a few years since the Boyd Neel string orchestra visited Australia under the auspices of the British Council. This provided more than a musical feast for those in the community who loved and understood music; it was a gesture of good will and of understanding from Britain to Australia. The time may well come, if indeed it has not already come, when Australia may be able to send to Britain some cultural organization which will bring Australia before the attention of the individual British people. This will be all to the advantage of Australia. Australia has sent to Great Britain more than one prominent medical investigator or man of science, and these have all helped to forge links which are of the utmost value in maintaining the integrity of the British Commonwealth. The present visit to Australia of her Queen is another link which will do far more than we can possibly imagine to weld the Commonwealth family together. The report of the British Council which has been discussed may appear at first sight to be as dry as dust, but it is really a document which contains much inspiration. It is most sincerely to be hoped that the British Government will see fit to restore to the Council the grant-in-aid which it felt compelled to take from it some twelve months or more ago.

THE MENTAL HOSPITALS SERVICE OF NEW SOUTH WALES.

For many years the annual report of the Inspector-General of Mental Hospitals of New South Wales has been the subject of comment in this journal. It is to be hoped that readers of the journal are not weary of reading about the deficiencies of this department. The report of the Inspector-General for the year ended June 30, 1952, was ordered to be printed on September 15, 1953. It has now been received. This journal would be failing in its duty if it did not draw attention to the deficiency of accommodation which, in this latest report, is stated to be increasing. The Inspector-General points out, as has been done before, that the deficiency in beds in mental hospitals is aggravated

by the arrival of thousands of people from overseas countries who have emigrated under various emigration schemes. Many of these people find their way into mental hospitals. In the report for the year 1950-1951 it was stated that the number of patients under care in the hospitals was greater than in any previous year, namely, 11,668. For the current year the number increased still further and on June 30 there were 11,964 patients in residence. The number of patients admitted for the year was 2308, which is the highest total so far reached. During 1950-1951 the number of patients in excess of accommodation was 2074; for the year 1951-1952 the number was 2213. The Inspector-General states again, as has also been stated before, that a building programme which is adequate to cope with the required accommodation has been approved by the Government and includes construction of two additional mental hospitals. Both projects have been authorized by an enabling Act of Parliament. However, no further progress can be reported in regard to either project, and sufficient money has been made available to proceed only with other constructional works which had already been commenced in the various hospitals. The details of these are set out. The new constructions will not provide any materially increased accommodation, but will provide for a higher standard than that existing previously, as they will replace old and unsuitable buildings. There are some other relatively minor works in progress which will give improved facilities for the treatment of patients. One of the satisfactory things about the report is that the numbers of the nursing staff recruited during the year have shown a considerable improvement, and in a number of hospitals the full staffs approved have been employed. The Inspector-General states that the clinical treatment of patients has been maintained during the year at a standard which can be regarded as satisfactory. He thinks that the nursing is comparable with that reached in other countries.

An interesting paragraph is included about the work at the Psychiatric Clinic at Broughton Hall. As is well known, the patients are admitted to this institution on a voluntary basis. The Inspector-General states that the advantages of this system are well known and the wisdom of extending it is fully recognized. He adds that, with this in mind, plans are being prepared to build on a site adjacent to Broughton Hall in order to provide further accommodation for the voluntary type of patient. This is something which will be seen possibly in the long-distant future.

Those who read successive reports of the Mental Hospitals Department of New South Wales will surely feel inclined to resort to sackcloth and ashes and to bemoan the ineptitude of the Government of New South Wales and its disregard for the mentally afflicted and their welfare.

Current Comment.

AXILLARY ODOUR.

MAN has probably always been aware of a distinctly unpleasant odour which emanates from his axilla. Since the axilla is remarkable, anatomically, for the presence of apocrine glands, the body odour from this area has been ascribed to this structure and the differences in odour in different races to varying numbers of apocrine glands.

The apocrine gland has then been considered to be a "scent" gland responding to emotional stimuli.

Little work has been done on pure, fresh apocrine secretion. Taking advantage of the fact that injections of adrenaline stimulate apocrine sweating without increasing eccrine sweating, W. B. Shelley, H. J. Hurley and A. C. Nichols¹ have made a detailed examination of apocrine secretion in the axilla. They have also studied to some extent the bacteriology of the normal axilla. Since the axilla is continually moist there is a large bacterial population. In order to obtain pure apocrine secretion the axilla was shaved, thoroughly cleaned with soap and water several times, then treated with 70% ethyl alcohol. When the last of the alcohol had evaporated, 0.15 millilitre of adrenaline one in 1000 solution was injected subcutaneously. The apocrine secretion was collected in sterile capillary tipped glass pipettes with cotton wool in the dilated distal end to insure sterility. On being tested for sterility a few of the tubes were found to be infected with common skin aerobic bacteria showing that the sterilization of the axillary skin surface was not perfect. Apocrine sweat, collected in this way, had no smell and did not develop any smell when kept at room temperature for fourteen days. Sweat collected from the opposite axillae of the same subjects, when the axilla was not shaven and not cleansed in any way, developed a strong odour in six hours which was very strong at twenty-four hours. Samples of the same sweat kept in the refrigerator developed no smell. It was to be expected that the smell would take longer to develop in the collecting tubes than in the axilla, if bacterial decomposition is the cause of the smell, for on the axillary skin there would be very many more bacteria than in the collecting tube. The addition of hexachlorophene, an antiseptic, to a tube of the sweat from an unprepared axilla prevented the development of smell for at least fourteen days. In order to determine what could be done to prevent the development of smell in the axilla itself the unshaven axillae of a number of subjects were washed daily for five minutes with a preparation containing hexachlorophene for varying numbers of days. There was a considerable decrease in the development of smell as compared with axillae treated with detergent only and there was a great reduction in the infected hairs. Shaving and careful, thorough washing of the axilla eliminate odour for more than twenty-four hours in most subjects. The use of a potent antibacterial substance gives even better results, but shaving seems to be essential for long-time protection. Soiled clothing can also contaminate apocrine sweat with development of smell.

Aluminium salts have been used considerably as axillary deodorants. In many subjects these are very effective, and the authors have shown that their action is mainly antibacterial but is also, in part, chemical on the odoriferous products. They do not prevent the secretion of apocrine sweat. Different odours from different people would appear to be due to variations in the types of bacteria predominating in the axilla. Pure eccrine sweat, whether sterile or unsterile, neither has nor develops an odour. Eccrine sweat, contaminated with sebum, keratin or debris, develops odour presumably as a result of bacterial action, but the odour is mild and quite distinct from that developing in apocrine sweat. Chlorophyll preparations could not be shown to have any axillary deodorant action when administered orally for a period of seven days.

INTERDIGITAL SINUSES OF BARBER'S HANDS.

ALTHOUGH very few cases have been reported in the literature, interdigital sinuses due to penetration of the skin of the hands and fingers are apparently common amongst barbers. A. R. Currie, T. Gibson and A. L. Goodall² were able to find reports of only 18 cases, and when their attention was directed to the lesion by a hairdresser reporting to the surgical out-patient depart-

¹ Arch. Dermat. & Syph., October, 1953.

² Brit. J. Surg., November, 1953.

ment at the Glasgow Royal Infirmary with an interdigital sinus of the hand, they could find no trace in the records of a similar case having been met with in the hospital. They were, therefore, surprised to find four other cases as a result of casual inquiry on their next visit to the hairdresser, and subsequently they examined the hands of 77 men's hairdressers from 18 different shops; of these, ten had the lesion in some stage of its development. They have also inspected the hands of 71 ladies' hairdressers and have not found one lesion of this type. Apparently, the lesion is usually regarded as too trivial to warrant the seeking of medical advice. The general story is that hairs tend to collect in the webs of the fingers of barbers during the course of their day's work, and not infrequently the short, sharp hairs from the heads of male customers may penetrate the epidermis and project from the skin surface. As a result of secondary infection in the superficial layers of the corium, small pits are formed just dorsal to the web margin, and more hairs gather in them with consequent persistence and aggravation of the reactive changes. Small sinuses are produced, and occasionally multiple sinuses develop in the same interdigital cleft. With the entry of more hairs through the sinus into the deeper layers of the corium and after further infective episodes, a longer sinus results. An abscess may form at its tip and point in the palm or dorsum of the hand, and it may rupture or be incised to produce a tunnel or fistula.

Discussing the factors determining the entry of hair at the site of the lesion, Currie, Gibson and Goodall point out that the only frequent site of formation of a chronic lesion appears to be the skin of the finger webs, most commonly between the forefinger and middle fingers, and between the middle and ring fingers of the right hand. Numerous theories have been suggested to account for the entry of hair into the skin, but these investigators believe that inadequate stress has been laid on the ability of short, sharp, clipped hairs to penetrate the epidermis in any situation, on the concentration of those hairs in the finger webs, and on the fine texture of the skin at this site. As a result of penetration of the epidermis by hair or hairs, secondary infection and foreign body giant cells are stimulated, and small pits are formed. More hairs readily accumulate in these, and the process continues with sinus formation. The entry of more hairs into the corium through the sinus opening may, perhaps, be aided by suction forces. Microscopic examination of sections of lesions excised showed that the sinuses were all lined by squamous epithelium, and even in those cases in which there was inflammation and suppuration at the sinus tip, there was little destruction of it. The epithelium apparently grows in a very regular fashion into the granulation tissue which precedes it, and in some cases the spinous cells appear to be in advance of the basal cells. This process continues as long as hairs are carried along the sinus to be lodged in the corium or subcutaneous tissue at its tip. If the entry of further hairs is prevented and the infection subsides, the opening at the sinus tip may become lined by epithelium to form a closed pit. Hairs may accumulate readily in a pit of this nature, and with penetration of the new epithelium by hairs and consequent reinfection, the lesion progresses and a tunnel is eventually produced. In a tunnel examined microscopically, there was evidence of chronic inflammation in the fibrous tissue around the epithelium, and on general principles Currie, Gibson and Goodall consider it reasonable to suggest that a squamous cell carcinoma might develop in such a lesion as a result of long-standing chronic irritation, although no such case has been recorded. They are very emphatic that the term "interdigital sinuses of barbers' hands" should be used to describe these lesions, and that the confusing term "pilonidal" should be avoided, not only for this particular lesion, but for those in certain other sites, namely, the umbilicus, the sole of the foot, the axilla and the supra-public region.

On the question of prevention and treatment, the view is expressed that if barbers paid sufficient attention to hand hygiene, and were careful to remove all the hairs

from the finger webs at the end of each day's work, this lesion could be prevented. It is rightly suggested that this should be brought to the notice of all barbers, as the condition is common and, although not of a severe nature, is an irritating disability. The treatment advised for the lesion depends upon the stage that it has reached. For lesions in the early shallow, pitted stage, excision was not advised or undertaken, because, if the patient removes all hairs from the web each night, it does not progress. Once the lesion has advanced beyond this stage, however, surgical excision is described as the treatment of choice, particularly if repeated infective episodes have occurred; care must be taken to remove any deeply seated granuloma. In Currie, Gibson and Goodall's series (they add 11 cases to those reported, bringing the total to 29) six of the patients advised to have surgical treatment accepted the advice. Two with multiple sinuses required a free skin graft after excision; in the remainder closure was effected by direct suture. Primary healing occurred in each case, and the results are described as satisfactory. However, the desirable and quite practicable approach to the condition is obviously that of prevention.

TUBERCULOSIS YESTERDAY AND TODAY.

TUBERCULOSIS is one of the oldest scourges of the human race. The pulmonary form was described by Hippocrates and spinal caries has been observed in the mummies of ancient Egypt. With the growth of large towns and cities it became more rife; only in our own day is civilization beginning to overcome this ancient enemy. In the seventeenth century Bunyan called it "the Captain of all these men of Death"; two hundred years later Dickens spoke of it as "the disease which medicine never cured, wealth never warded off". For hundreds of years it has been accepted with a sense of despair and resignation as an unavoidable disease. During the nineteenth century "galloping consumption" was a personal experience in every group in the community. According to René J. Dubos,¹ three discoveries changed this attitude to one of hope and combat. During the latter part of the nineteenth century, humanitarians and sociologists noticed that tuberculosis flourished among the poor and overcrowded, and that consequently some measure of control against the epidemic could be achieved by social reforms. Villemin in 1868 had pointed out that tuberculosis was uncommon in mountain areas and that it spared people who lived in small communities, and in the open air. It was found that prolonged rest and good food in pleasant airy surroundings could help the stricken individual to overcome his disease or at least to live in relative comfort with it. This led to the development of sanatoria and encouraged the hope that the physiological factors affecting resistance to tuberculosis would soon be defined in precise terms. Finally the discovery of the tubercle bacillus by Koch in 1882 dispelled the mystery which had till then surrounded the disease. This new knowledge and the hope which it brought soon turned passive and resigned compassion into a fighting faith: the antituberculosis movement grew up in this atmosphere and united the social reformers, the hygienists and the bacteriologists in a crusade.

In an address made at the Fiftieth Anniversary Meeting of the New York Tuberculosis Association, Dubos said:

Statistics were not necessary to alert the public to the importance of tuberculosis when the Association was founded fifty years ago. At that time, the cough of the tuberculous was heard everywhere in our communities; a feverish glow betrayed the advanced stage of consumption in two out of every hundred persons and foretold of their death within a few years. Tuberculosis was then the White Plague, the greatest single cause of disease, destruction, and death in Western Europe and in North America. It found its victims among young men and women in their prime.

Today, he pointed out, the antituberculosis movement works in a very different atmosphere.

¹ Am. Rev. Tuberc., July, 1953.

The cough of the tuberculous is no longer heard in public places. Most laymen and even some physicians are becoming unfamiliar with the symptoms, sufferings and despair of victims of tuberculosis. This is due in large part to the immense progress made during the past fifty years in the conquest of the disease, as witnessed by the spectacular decrease in its mortality. The annual tuberculosis mortality in Greater New York was 242 per 100,000 population in 1902; it will not be much higher than 21 in 1952—better than a tenfold decrease in a period of fifty years. But this spectacular triumph over death, which we so justifiably advertise, is clouded by our failure to prevent disease. . . . If the cough and fever of tuberculosis are no longer haunting our cities, it is, in part at least, because a large percentage of tuberculosis patients are now segregated in specialized hospitals and sanatoriums where they live a crippled existence on the margin of normal society.

In England, as in the United States, reduction of mortality following the extensive use of antibiotics and of surgery is not matched by reduction in morbidity. G. L. Cox and H. F. Hughes¹ insist that a new measure of progress in the fight against tubercle is needed, for death rates give a false impression of progress. What is needed in Britain is a "live" register of infectious cases for the use of local health authorities. Only by wider and greater use of public health measures to discover and control sources of infection can the great undiscovered reservoirs of active infectious cases be reduced and finally eliminated. Dubos agrees with these writers that drugs or surgery rarely bring about a complete cure of tuberculosis. This fact, he says, is of enormous practical importance and is not sufficiently well known. It is not realized by the laity; in particular it is not appreciated by the average patient with chronic tuberculosis. These patients constitute our most dangerous, because least known, reservoir of infection at the present day. That they are so is the outcome of pathological characteristics peculiar to tuberculous infection; it is due to the prolonged survival of bacilli in caseous lesions where they are out of reach of the normal defence mechanisms of the body and also of antimicrobial drugs. This accounts for the recurrence of disease in the tuberculous individual and for the persistence of what Dubos calls a "huge reservoir of infection in the community". Today men of middle age form the most important reservoir of tuberculous infection, and these are a group least readily approached by screening programmes. Human infection is a far greater problem than bovine infection; indeed Dubos states that bovine tuberculosis has been practically (though not entirely) wiped out from the United States of America. When B.C.G. vaccination was first used it was hoped that this would help to break the chain by rendering contacts immune to tuberculosis. Unfortunately the immunity induced by B.C.G. vaccine is at best partial and, according to Dubos, it certainly does not prevent virulent tubercle bacilli from getting a foothold in the body. He believes that efforts aimed at increasing the general resistance of the body would contribute to progress, not only in the control of tuberculosis, but also in that of several other infectious diseases as well; it is, he states, a remarkable fact that the death rate of several bacterial respiratory infections follows a course parallel to that of tuberculosis. Unfortunately we really know very little about general resistance to infections; the factors which control it need intensive and original research. No one can doubt the importance of such research in the prevention of tuberculosis; Dubos states that:

The inner logic of the programme of antituberculosis associations should lead them to become truly health associations. For it can hardly be doubted that, in the final analysis, health is a positive attribute rather than merely the absence of disease.

This emphasis on general health is all the more impressive since it comes from a great bacteriologist. In concluding his address, he said:

Each generation has its social responsibilities. Ours are harder to perceive because, unlike our ancestors, we have little occasion to experience contact with

hosts of people dying of obvious tuberculosis in the midst of our cities. The generous emotions which are the motivating power of all creative social enterprise must now be generated from the knowledge of cold statistics instead of through the blood-stirring effect of direct experience. But cold statistics can also be eloquent. They tell of thousands of hospital beds, of hundreds of millions of dollars paid out of public funds, of even greater economic losses caused by decreased productivity due to tuberculosis. Even more, they call to mind many thousands of our fellow men living a crippled existence in silent despair, with broken homes, shattered hopes, potentialities unfilled.

All this can be prevented, for it is known that societies have existed and still exist almost completely free from tuberculosis. This was the happy fate of many primitive people before the white man came to disturb the wisdom of their ancestral way of life. More important still from our point of view is the fact that there are today communities in the industrialized Western World which were once riddled with tuberculosis and which have now all but eradicated the disease. Thus, it is certain that tuberculosis is not an inescapable component of human society. It is always the result of gross defects in social organization and in the management of individual life. It is truly a social sin which can and must be stamped out.

This kind of utterance should spur the National Association for the Prevention of Tuberculosis in Australia (NAPTA) to further effort and medical practitioners and others to support it.

THE INEFFECTIVENESS OF EXPECTORANTS.

The chronic cough remains one of the commonest conditions in general practice and the prescription of an expectorant mixture an almost invariable consequence. There has been much in medical literature suggesting that this or that so-called expectorant has little or no effect. Ian Rose has examined the effects of a number of the common expectorants on patients in relation, in particular, to (a) increase or diminution of sputum; (b) the viscosity of the sputum; (c) increase or decrease of cough; (d) the time relation between the administration of the expectorant and any change noted.

It is very improbable that ammonium chloride and ammonium carbonate have any direct action in the bronchi, for on absorption they are immediately converted into urea and it is extremely difficult to raise the ammonium content of the systemic blood from the normal very low figure. Any effect these substances might have could possibly be due to gastric irritation causing a reflex bronchial secretion. In Rose's series neither drug had any effect on the sputum or on the cough. There is no physiological basis for the use of citrates and acetates as expectorants. They are very rapidly metabolized in the body after absorption. They were without any apparent effect on the cough or sputum. The iodide ion is said to be excreted by the bronchial glands and to "loosen" a cough. As others have done, the author found no change in the sputum after giving iodides, but there was some subjective relief of the dry cough. There was no apparent effect with ipecacuanha. Creosote is not excreted in the lungs nor is any derivative of it. Beyond causing a brief stimulation of the cough immediately after it was taken, creosote was not found to have any effect. Guaiacol, too, was found to have no effect. Terpene hydrate in the usual doses had no effect, but when five times the usual dose was given there was some "loosening" action. Opium alkaloids, particularly codeine, were useful in quietening a cough.

When any of the expectorants mentioned were given in a glass of water or milk they produced a fairly consistent loosening of the cough and a slight increase in the amount of sputum in the half-hour following. However, this response was equally apparent with a glass of salt and water. A mixture of sodium bicarbonate, sodium chloride, chloroform emulsion and anise water gave as good results as any of the expectorants alone or in mixture.

¹ *Tubercle*, September, 1953.

² *Canad. M. A. J.*, September, 1953.

Abstracts from Medical Literature.

SURGERY.

Duodenal Diverticula.

A. CHITAMBAR (*Surgery*, May, 1953) reviews the literature on duodenal diverticula, adding cases of his own and discussing the subject from many aspects. He stresses the frequency of the condition—5.76% in his series, diagnosed radiologically—with a slightly higher incidence in males, and increasing in frequency up to the fifth decade of life. The majority of cases occur in the second part of the duodenum. The author discusses various viewpoints of aetiology and describes in detail the pathological changes and complications which occur. He lists as symptoms pain, constipation, flatulence, nausea, vomiting and loss of weight. The treatment is usually medical—diet, postural drainage of the diverticulum and the use of antispasmodics. Surgery is indicated if inflammation or neoplasm occurs, or if there are symptoms from stasis within the diverticulum or from obstruction to biliary tract, pancreas or duodenum. The difficulties and dangers of surgery are described. In conclusion, the author states that the greater number of diverticula do not cause symptoms, but a small percentage need active therapy.

Carcinoma of Rectum and Sigmoid.

V. C. WAITE (*Surgery*, May, 1953) discusses the method of spread of carcinoma of the rectum and sigmoid with special reference to the anatomical considerations of lymphatic drainage of the area. He stresses that upward lymphatic extension leads to involvement of nodes in the vicinity of origin of the left colic artery. As a result he recommends more radical upward resection for carcinoma, with removal of the inferior mesenteric artery at its aortic origin and the inferior mesenteric vein at its highest level. This necessitates terminal colostomy of the descending colon in abdomino-perineal resection of rectum. With abdominal resection and anastomosis the splenic flexure is mobilized, and the descending colon is brought down to the pelvis to join with rectal remnant.

Cysts of Thyroglossal Duct.

C. E. REES AND MAURICE J. BROWN (*Am. J. Surg.*, May, 1953) discuss the embryology and pathology of cysts of the thyroglossal duct. They stress that the thyroglossal tube passes through or behind the body of the hyoid bone. They state that 79% of cysts occur in the mid-line and 21% laterally. Removal following the technique of Sistrunk is advised. The authors describe 18 cases so treated with no recurrence or complication following surgery.

Renal Injuries.

W. F. SURREMONT AND J. W. LIDENS (*Arch. chir. neerl.*, Vol. 5, Feb. 1, 1953) present a follow-up study of 57 patients with renal injuries, who had been conservatively treated. The patients had been followed for periods of one to twenty years. One patient had had an infected kidney and then developed a

low degree of hydronephrosis without infection; none of the patients had developed hypertension. The authors conclude that the only indication for active treatment in retroperitoneal renal injury is increasing pain and tenderness in the flank with clinical symptoms of severe haemorrhage. All other minor and major injuries should be treated conservatively. Late disturbances in such damaged kidneys are minimal.

The Roux Loop.

P. R. ALLISON AND L. TRAVARES DA SILVA (*Brit. J. Surg.*, September, 1953) state that experience of hiatal hernia and reflux peptic oesophagitis has thrown more and more doubt on the wisdom of performing mediastinal oesophago-gastric anastomoses. In their unit the operation has been virtually abandoned for many years, except for its occasional use as a purely palliative measure in a patient with a very short expectation of life. In its place oesophago-jejunal anastomoses have been used. The technique for the preparation of the Roux loop is described, and results are given to show that with care and experience its use need not seriously affect the operative mortality. The jejunum is divided four inches from the duodeno-jejunal junction, and the distal loop is brought straight up to the point of anastomosis without tension and without surplus length. The estimate of the required length of loop should be made at the beginning of the operation, for the risks are increased if vessels are divided unnecessarily to make too long a loop, or if a good loop is stretched only a centimetre too far and its draining vein occluded. The end of the distal jejunal loop is closed early in the operation and dropped back into the abdomen until it is needed; after this interval any doubts about its viability can be answered. Later, the oesophageal anastomosis is made in the immediate subterminal part of this loop. Continuity is restored by a T-anastomosis of the short proximal jejunal loop to the left side of the distal loop. Whenever possible, the loop is placed in a retroperitoneal position. In a series of 28 simple lesions at or about the cardia there has been no operative complication referable to the Roux loop, and in 61 operations for malignant lesions there were eight deaths which might have been associated with the use of the Roux technique. In three of these the loop became gangrenous, and in five there was a faulty mediastinal anastomosis.

Bleeding Peptic Ulcer.

H. C. SALTZSTEIN, M. S. SAHLIN AND S. R. SCHEINBERG (*Arch. Surg.*, July, 1953) review 402 cases of massive upper gastro-intestinal haemorrhage, but they point out that each case must still be considered as an individual problem. In their series gastric ulcer occurred one-fifth as frequently as duodenal ulcer, but deaths from gastric ulcer were almost three times as frequent as those from duodenal ulcer. Of the 402 cases the site of the bleeding was not discovered in 59. (Oesophageal varices and carcinoma were excluded.) Of the remaining 343 patients 80% were treated medically and the other 20% surgically. The medical mortality was 5.1% and the surgical 5.9%. The need to avert the ill effects of anoxia on the

liver is stressed. It has been shown that bleeding from the gastro-duodenal artery will cause the blood pressure in the hepatic artery to fall much more rapidly than will the loss of the same amount of blood from the peripheral circulation. This selective reduction of blood flow is greatest when the systemic blood pressure reaches levels below 100 millimetres of mercury. Anoxia can be produced very easily in the liver. Although 75% of the blood supply of the liver is derived from the portal vein, 50% of its oxygen supply comes from the hepatic artery. Even in early shock, the portal vein is quickly drained of its oxygen saturation (which is normally 20% less than that of arterial blood), and the liver is then dependent upon the hepatic artery blood flow for its entire oxygen supply.

Vital Staining of Lymphatics in Colon Surgery.

J. WEINBERG AND H. J. MOVIUS (*West. J. Surg.*, September, 1953) report further the technique of injection of the lymphatics near a carcinoma of the colon at the time of operation. As soon as the abdomen is opened three to five mls of a 4% solution of direct sky blue in distilled water plus not more than six units of hyaluronidase are injected into the intestinal wall immediately proximal and distal to the cancer. The injection is made with a 26-gauge needle attached to a Luer-Lok syringe. The vivid blue colour is taken up by the regional lymphatics and is retained for some hours. Within fifteen minutes the lymphatic nodes should be visible, even through thick layers of adipose tissue, but certain conditions interfere with the uptake of the dye. Extension of cancerous tissue into and around the lymphatics close to the primary lesion may act as a complete block. If only part of a node is occupied by cancer, the normal tissue within the node will receive the stain while the cancer remains unstained. Apart from the use at operation in determining the extent of the local lymphatic field, this method also facilitates the recognition by the pathologist of the lymphatic nodes in the specimen after its removal.

Chronic Lymphoedema of the Extremities.

J. WATSON (*Brit. J. Surg.*, July, 1953) surveys the conditions causing chronic lymphoedema of the extremities. In addition, its anatomical, physiological and pathological aspects are considered. The following are the main types: (i) Milroy's congenital lymphoedema, which presents as a smooth uniform enlargement of the limb at birth. It is very rare and arises as both a congenital and hereditary affection. The initial lesion appears to be a lymphangectasis of the limb. (ii) *Lymphoedema praecox*, which is the commonest type in this series. Its usual onset is in the second or third decade and it usually affects one lower limb. Pain is usually absent, and ulceration is rare. (iii) Infective lymphoedema, which is due either to filaria or to non-specific infections. In the latter cases progression is aggravated by a series of inflammatory episodes often associated with severe constitutional disturbance. (iv) Secondary obstruction in relation to malignant disease or following operation. (v) Trauma, which is usually followed by lymphoedema of the limb distal to circumferential burns or stripping of the skin and subcutaneous tissue. (vi)

Reflux chylous lymphoedema due to lymphatic obstruction either of the thoracic duct itself or at the root of the mesenteries. The numerous surgical procedures described for treatment include the following: (i) Attempted reconstruction of the lymphatic channels by the introduction of silk threads or buried rubber tubes; but a case is described in which silk threads had been buried for twenty years without relief. On removal these were found to be heavily calcified and rigid rods, obviously without any capillary action. Reconstruction may also be by the transplantation of a living "lymphatic bridge" consisting of skin, fat and correctly orientated lymphatics to bypass inguinal or axillary obstruction. The further development of this method is said to offer hope of providing a radical cure for the disease. (ii) Sympathectomy, which is not advised. (iii) Attempted drainage of the stagnant pool into the muscle compartments of the limb by excision of strips of deep fascia; but this assumes the patency of the deep channels, which is doubtful. (iv) Resection of the skin and subcutaneous tissues, and the application of a Wolfe graft to the raw surface. The details of this method are given as this is the method recommended. It was employed in 16 out of 26 cases with good results in 13.

Idiopathic Segmental Haemorrhagic Infarction of Greater Omentum.

RAYMOND J. TILLY, JUNIOR (*Ann. Surg.*, August, 1953), describes a case of idiopathic segmental hemorrhagic infarction of the greater omentum and tabulates in a summary the cases reported in the literature. It would appear that the condition usually occurs in well-nourished, frequently obese individuals. It is seen more frequently in males. Usually, the pre-operative provisional diagnosis has been acute appendicitis, although perforated ulcer and acute cholecystitis both appear as the provisional diagnosis in the summary of cases. The author discusses and reviews the possible aetiological factors, such as the amount of fat in the greater omentum and whether the patient had partaken of a meal immediately before the onset of the pain. The treatment is wide excision of the infarcted segment. With this the prognosis is quite good; all but one of the patients reviewed recovered.

Prevascular Femoral Hernia.

D. P. B. TURNER (*Brit. J. Surg.*, July, 1953) reports a case of prevascular femoral hernia and points out that only five cases of this type of hernia have been described in the English and American literature. A prevascular femoral hernia is described as one in which the sac bursts through the fascia transversalis onto the anterior aspect of the femoral vessels. It is then situated within the femoral sheath and extends laterally across the front of the iliac and femoral vessels. This does not include femoral herniae which have traversed the femoral canal and then deviated laterally across the front of the femoral vessels. In the case reported here the neck of the sac accommodated four fingers placed side by side and extended from Gimbernat's ligament to about an inch lateral to the femoral artery. After mobilization and excision of the sac the large defect was repaired with "Nylon" sutures

placed at intervals of one centimetre through the ilio-pectineal ligament (extending from the pubic spine to close to the femoral vein), the conjoined tendon and the inguinal ligament. In addition, the very lax inguinal ligament was strengthened by a strip of pectineus muscle fascia, which was raised from the muscle medial to the vessels and carried across in front of them to be stitched, at a point an inch lateral to the external iliac artery, to the inguinal ligament in front and to the ilio-psoas fascia behind. After relief incisions had been made in the deep layer of the rectus sheath, the external oblique was closed without tension.

Celiac Artery Ligations in Portal Cirrhosis.

W. K. JENNINGS and J. P. BLANCHARD (*West. J. Surg.*, October, 1953) summarize their experience with ligations of the major divisions of the celiac artery in seven patients suffering from portal cirrhosis. They state that insufficient time has passed to permit accurate appraisal of the effectiveness of hepatic, splenic and left gastric artery ligations in the treatment of portal cirrhosis, but two of their seven patients are alive and free of symptoms twenty-four and six months, respectively, after such surgery. It is advised that ligation of the hepatic, splenic and left gastric arteries should be accomplished through the omental bursa with the stomach rotated upwards, and that a careful search should be made for anomalous, or accessory, hepatic arteries. In the light of present knowledge it appears that active bleeding, continued jaundice and an enlarged liver should be regarded as contraindications to these arterial ligations. Observations to date suggest that the patient with an atrophic liver and well-established collateral circulation may expect to derive the greatest benefit from them. Their series is too small to warrant any conclusions regarding the relationship of liver size to the probable outcome of the ligation procedure; but, in general, the results tend to support the opinion that an enlarged liver is a contraindication. However, because of the difficulty in estimating the size of the liver, it is possible that prognosis would be better based on biopsies of the liver if a series of such biopsies were obtained and were correlated with the subsequent post-operative courses.

Strangulated Hiatus Hernia.

G. A. P. HURLEY (*Ann. Surg.*, August, 1953) states that up till quite recently the opinion was widely held that herniation of the stomach through the oesophageal hiatus of the diaphragm was a relatively benign condition, well suited to medical management. It has always been admitted that hiatus hernia involving intestine was liable to strangulation and constituted, therefore, an indication for surgical intervention. The author presents the notes of two cases of herniation of the stomach through the oesophageal hiatus which constituted an urgent indication for operation. In one of the cases part of the stomach wall was gangrenous and had to be resected. The author states that from the practical point of view, one must try (i) to recognize the cases of hiatus hernia of the stomach in which strangulation may occur, (ii) to diagnose strangulated hiatus hernia when it occurs and (iii) to have a plan

of treatment. Strangulation must be envisaged as a likely possibility when (a) there is a large amount, say one-third, of the stomach incarcerated in the hernia, and when X-ray examination shows constriction of the stomach by a relatively small hiatal opening, (b) the hernia has appeared or greatly increased in size at the time of, or shortly after, a crushing injury of the abdomen or lower part of the chest, and (c) the stomach has undergone inversion or torsion, with the greater curvature lying high in the chest.

Sterility of Surgical Linen.

H. D. PROBST (*Am. J. Surg.*, September, 1953) points out that previous research in wound contamination has been focused primarily on the operative site, surgeons' hands, masks, instruments, autoclaves, air-borne contamination and suture material, and that little attention has been given to the sterility of surgical linen during operation, except to avoid contamination by direct contact with unsterile objects. However, once cloth has become damp, even if the water source is sterile, it allows contamination to take place provided it is in contact with an unsterile object. Linen which has become dampened by perspiration is, of course, already contaminated. In order to find a useful method of eliminating contamination through dampened linen, bactericidal substances, such as "Roccal", were added to the linen, and cultures were made to determine the dilution which would prevent such bacterial migration. In the case of "Roccal" a dilution of 1:2500 was found to be effective, and in this dilution no untoward effect was noticed.

Total Right Hepatic Lobectomy.

GEORGE T. PACK and HARRY W. BAKER (*Ann. Surg.*, August, 1953) made a pre-operative diagnosis of hepatoma of the right lobe of the liver in a forty-year-old man who had been sick for one year. Under intratracheal cyclopropane anaesthesia, the abdomen was first explored through a right upper rectus muscle-splitting incision. The abdominal incision was extended across the costal margin obliquely, and the right side of the chest was entered. A complete right hepatectomy was performed with success and the patient discharged from hospital thirty-six days after the operation. The specimen, which is described microscopically, has been called plasma cell granuloma. The nature of the infection which was responsible for this granulomatous condition remained obscure, despite careful investigation.

Wire Sutures and Ligatures.

J. I. KNOTT (*Am. J. Surg.*, August, 1953) discusses the virtues of stainless steel wire as a suture and as a ligature material and gives in detail various points in the technique of its use. Fine steel wire is said to be a reliable suture and ligature material for hernia repair and for wound closure. The proper handling of fine wire overcomes most of its limitations, and such handling is facilitated by (i) using short 14-inch pre-cut strands, which can be handled without kinking, (ii) double threading the needle to reduce tissue drag at the needle eye, (iii) making ties across the finger tips of the left hand with the needle holder, (iv) placing stitches with the assistant holding the tissues ahead of the needle, and (v) using the finer sizes of wire.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XCIV.

CORONARY ARTERY DISEASE.

DISEASE of the coronary arteries is usually due to atherosclerosis. On this a great deal of research is still being done. Probably the main factor in its development is a disturbance of metabolism causing a deposit of lipid substances in the internal coat of the blood vessels, and lesions similar to atherosclerosis have been produced experimentally in animals by feeding with cholesterol. Also, diseases such as *diabetes mellitus*, nephrosis, myxœdema, obesity and hereditary lipodosis are frequently associated with atherosclerosis.

It is interesting that women, before the menopause, are much less liable to coronary artery disease than men, and it is believed by many workers that the oestrogenic hormone gives women a considerable degree of immunity to coronary atherosclerosis.

The earliest lesions are seen as yellowish linear streaks of lipid material in the deeper layers of the internal lining of the blood vessels. These streaks gradually increase in size, encroaching on the lumen. Sometimes they break through and ulcerate, and upon this rough surface clotting frequently takes place. Calcium salts may be deposited in the lipid material, and the vessel becomes hard and brittle. The result of these pathological processes is that the wall thickens, the lumen narrows, and in some cases the vessel becomes completely obstructed.

As the vessel narrows, ischemia may cause some degree of fibrosis of the cardiac muscle, but in many cases there is no structural change.

Complete obstruction causes infarction of portion of the cardiac wall, and this is followed by fibrotic infiltration to repair the damage. If the pericardium is involved local pericarditis follows; if the endocardium is involved, mural thrombi may form on the necrosed area, or, more rarely, the heart muscle may rupture.

The vessel most commonly involved is the anterior descending branch of the left coronary, less frequently the right coronary, and the left circumflex artery.

The usual site of infarction is the left ventricle, generally the anterior apical portion—sometimes the posterior wall.

Infarction of the wall of the left ventricle frequently extends to the interventricular septum, and may involve the right ventricle. Right ventricular infarction rarely occurs alone.

Angina Pectoris.

Angina pectoris is usually caused by atherosclerosis of the coronary vessels resulting in myocardial ischemia. Sometimes it is due to aortic stenosis, syphilitic aortitis or the sudden onset of auricular flutter or tachycardia. Severe anemia is occasionally a contributory factor. The attack is usually precipitated by emotional disturbances or physical strain, and is particularly liable to occur after meals and when the patient is walking up hill or against a cold wind. The pain is substernal and radiates to one or both arms, sometimes to the neck or jaw. It is constricting or crushing in character, is continuous, but lasts only a few minutes, and occurs immediately after exercise. It is rapidly relieved by rest, and reappears if exercise is again attempted.

Physical examination findings may be entirely negative unless hypertension or aortic stenosis is present. Occasionally protodiastolic gallop rhythm is heard.

The electrocardiogram is usually normal unless myocardial ischemia has given rise to branch bundle block or hypertension is present with cardiac hypertrophy. Exercise to the point where pain is produced will often cause a temporary S-T deviation, but it must be remembered that exercise is not altogether without risk.

Differential Diagnosis.

Anxiety states and intercostal neuralgia cause the greatest difficulty. In these the patient complains of a pain in the left mammary or inframammary region, usually sharp or stabbing in character, not continuous and not directly related to exercise. It often lasts for hours and not for a

few minutes, as does true angina. These symptoms will establish the diagnosis in most cases, but occasionally there may be doubt, and an electrocardiographic tracing before and after exercise may be necessary.

Hiatus hernia usually causes a burning substernal pain, closely resembling *angina pectoris*, but the pain is related to the intake of food and not to exercise and does not have the typical radiation of angina. A barium meal and electrocardiographic examination will usually make the differentiation possible.

Spinal root pain is generally bilateral and not related to exertion.

Treatment.

A patient who suffers from attacks of *angina pectoris* must endeavour to avoid any exercise or emotion which produces pain. He should be particularly careful to avoid exercise after meals. A long holiday and change in restful surroundings are often beneficial.

Excessive smoking should be avoided and obesity treated by dietary restrictions. Alcohol may be taken in moderation and in some cases will relieve pain.

Tablets of trinitrin, grain 1/100, are usually effective and are now much more commonly used than inhalations of amyl nitrite.

Aminophylline, grains one and a half to three, three-times daily, is often given, but the benefit obtained is doubtful.

More recently khellin, 100 milligrammes three times daily, has been recommended, but its value has still to be proved.

If the pain is severe and occurs on very slight exertion, complete rest in bed for three or four weeks will often relieve the patient's symptoms. In cases of intractable and crippling pain, injection of alcohol into the dorsal ganglia has been recommended, but the number of patients who require this treatment is very limited.

If the pain of angina becomes acute and prolonged, anti-coagulants should be given immediately to prevent the onset of myocardial infarction.

Coronary Thrombosis and Myocardial Infarction.

In cases of acute coronary artery obstruction the patient is suddenly stricken with an intense vice-like substernal pain, which extends across the chest to one or both arms or the jaw. It lasts for hours and is accompanied by pallor, sweating and collapse. Dyspnea varies from slight breathlessness to the acute distress of cardiac asthma or pulmonary œdema.

If the coronary obstruction develops slowly, the infarction may be completely "silent", and the detachment of a mural thrombus followed by embolism in a peripheral vessel may be the first sign.

When the obstruction is acute, the blood pressure usually falls, and may never return to its former level. The heart sounds are rapid and soft, and a proto-diastolic gallop rhythm may be present.

Transient pericardial friction may appear on the second or third day and is a valuable diagnostic sign.

Extrasystolic contractions frequently occur and may herald the onset of ventricular tachycardia or fibrillation.

The temperature is usually raised for a few days and the sedimentation rate increased.

In most cases an electrocardiogram will enable an immediate and certain diagnosis to be made. In the early stages S-T deviation appears above or below the iso-electric line, followed by characteristic alterations in the contour of the T waves, and in some cases by the appearance of deep and broad Q waves, which are pathognomonic of myocardial infarction. However, there may be no immediate change in the electrocardiogram, and confirmation of the clinical diagnosis can be made only by serial tracings taken preferably two, four, seven and fourteen days after the infarct. Characteristic changes will almost invariably be found in the serial tracings and will establish the diagnosis.

Complications.

Ventricular fibrillation causes approximately 10% of the deaths following acute myocardial infarction.

Auricular flutter and fibrillation frequently occur and may be mistaken for ventricular fibrillation. Even with the electrocardiogram the diagnosis may be difficult if, in

addition to the disturbance of auricular rhythm, there is also aberrant ventricular conduction.

Branch bundle block is found in about 10% of cases.

Auriculo-ventricular block is less common. If it is incomplete, and the degree of block varies, the rhythm may be irregular. It is then difficult to distinguish this condition from extrasystolic contractions until an electrocardiogram is taken.

Congestive cardiac failure may follow auricular flutter, fibrillation or left ventricular failure.

Ventricular aneurysm usually follows a large infarction, and radiographically may be seen as a localized bulge in the ventricular wall. A Q wave is usually present in the electrocardiogram and a persistent elevation of the S-T segment with a small R in lead I and a deep S in leads II and III.

Cardiac rupture, a rare complication, causes haemopericardium and death—immediately or in a few days. It usually follows hypertension or early ambulation after a large infarction.

Thrombo-embolism has caused a great deal of interest and discussion in recent years and is found in 10% of cases. It occurs between the fourth day and the fourth week after the infarction. Thrombosis occurs most frequently in the veins of the legs and may give rise to emboli, which cause massive infarction of the lungs and sudden death. Mural thrombi which develop upon the myocardial infarct and become detached give rise to emboli in the renal, splenic, cerebral, mesenteric and peripheral arteries.

Differential Diagnosis.

With pulmonary embolism, the symptoms, signs and electrocardiographic appearances in the standard three leads closely resemble those with posterior myocardial infarction, but the chest leads will differentiate between the two conditions.

Although the symptoms of dissecting aneurysm resemble those of myocardial infarction, the pain in the former usually extends to the back and often to the legs. An aortic regurgitant murmur is heard, the carotid, renal or femoral arteries may be cut off, and the electrocardiogram is normal.

Acute pericarditis, cholelithiasis and perforated peptic ulcer have been diagnosed as myocardial infarction, but the situation of the pain is abdominal rather than thoracic, the distribution is different, and the electrocardiogram is normal.

Hiatus hernia is distinguished by the radiographic appearances and the normal electrocardiogram.

Prognosis.

The committee set up by the American Heart Association states that the immediate mortality rate is 15% among patients treated with anticoagulants and 23% among those not so treated.

It is difficult to estimate the life expectancy of the individual who recovers from the acute phase of myocardial infarction. I believe that on the average his remaining years are halved.

Treatment.

If the patient is very severely shocked and the blood pressure is low, complete rest in bed in the recumbent position is necessary. For the first few days he must not feed himself, and continuous nursing is required.

If not severely shocked he should be propped up and allowed to move his limbs, feed himself and use a commode rather than a bed-pan. Complete immobilization has a bad psychological effect upon the individual, and predisposes to thrombo-embolic complications, just as it does after surgical operations.

Morphine is essential in the early acute phase of the disease.

Oxygen should be given if the patient is cyanosed.

Visitors should be restricted in numbers and especially in the time they are allowed to stay with the patient.

If frequent extrasystolic contractions are present, quinidine sulphate, grains three to six, or procaine amide, grains seven and a half, is given every four hours to prevent the onset of ventricular fibrillation.

If auricular flutter, fibrillation or congestive cardiac failure develops, digitalis should be given, notwithstanding the statement frequently made that the administration of digitalis is contraindicated in cases of myocardial infarction. The

amount recommended is digitoxin 1.2 milligrammes or digoxin 2.0 milligrammes in twenty-four hours, followed by a daily maintenance dose of 0.2 milligramme of digitoxin or 0.5 milligramme of digoxin.

The administration of digitalis in these conditions appears to be safer than that of quinidine, particularly in cases of auricular flutter, when administration of quinidine may be followed by a rapid ventricular rate to 1:1 conduction.

In cases of congestive failure salt should be restricted, and intramuscular injections of one to two millilitres of mersalyl given until the oedema disappears.

No special treatment is required for the irregular rhythm which sometimes occurs in partial or incomplete auriculo-ventricular block, but quinidine is contraindicated.

Blood transfusion followed by the subcutaneous injection of norephedrine has been recommended by some physicians in cases of severe shock, to combat the decline in coronary and peripheral circulation, but patients suitable for this treatment are very rare.

Thrombo-embolic episodes occur in 10% of all cases of myocardial infarction, and anticoagulant therapy has undoubtedly reduced the mortality rate. Considerable discussion has taken place in recent years on whether this therapy should be reserved for the "bad risk" subjects, who suffer from severe shock, intractable pain or evidence of cardiac failure. It has been contended that the "good risk" subjects, comprising up to 40% of the total, do not require anticoagulants, because in this group the mortality rate is low, and it has been said that the number of thrombo-embolic complications is small. However, I have met with several instances of peripheral embolism in patients who have had a "silent" infarction and the diagnosis was made only after a routine electrocardiographic examination. The risk of venous thrombosis in the lower extremities and of pulmonary embolism is certainly greater in the "bad risk" subjects who are suffering from severe shock and are almost completely immobilized. But the possibilities of embolism from a mural thrombus are present in both "good risk" and "bad risk" subjects.

Anticoagulants should therefore be given in all cases of myocardial infarction if it is practicable, and in particular in the severe cases and in those in which cardiac aneurysm has developed.

It is essential that the administration of anticoagulants should be controlled by daily estimation of the time required for citrated or oxalated blood to clot after recalcification. This is the prothrombin time.

The prothrombin time of a sample of plasma from a normal control is estimated at the same time as the patient, and the ratio of the two is known as the prothrombin index—a control time of twelve seconds and a patient's time of twenty-four seconds would give a prothrombin index of 50%.

The prothrombin index should be maintained at 35% to 55%. If it is more than 55% the treatment is useless, if less than 35% there is danger of haemorrhage.

And so, unless a trained pathologist is available, and the patient can afford his services or be admitted to a public hospital, anticoagulant therapy cannot be undertaken. In this country, where many people live in remote areas, and others are unable to afford the treatment and cannot obtain admission to a public hospital, the number of people receiving anticoagulant therapy must necessarily be limited. This is regrettable, as the death rate is undoubtedly lowered by this treatment, and the risks are minimal if the administration is well controlled.

The anticoagulants most commonly used are dicoumarol and "Tromexan".

On the first day 300 milligrammes of dicoumarol are given and on the second day 200 milligrammes; and the dose is then tapered off to 100, 75 or 50 milligrammes, according to the prothrombin time. As the full effect of dicoumarol is not felt for three days, the newer and more rapidly acting anticoagulants, such as "Tromexan", are often used.

The dose of "Tromexan" is four to five times that of dicoumarol—that is, 1200 to 1500 milligrammes on the first day, 800 to 1000 milligrammes on the second day, and a maintenance dose of 250 to 400 milligrammes daily.

"Tromexan" acts in twenty-four hours, but it is slightly more variable in its effects than dicoumarol, and it should be given at the same time each day, and as soon after the prothrombin test as possible. In severe cases, if dicoumarol is being used, heparin is given in doses of 5000 units every four hours for thirty-six hours. If "Tromexan" is used, heparin is not generally required.

As recent researches appear to have established a relationship between the fat content of the diet and atherosclerosis, it is desirable to restrict the total intake of fat—margarine and vegetable oils, lard, dripping and fatty meats, bacon and pastry. Eggs and dairy products such as milk, cream and butter should be taken only in limited quantities. Obese patients must, in addition, be given a diet of low Calorie content and their weight reduced to normal limits.

Although the effect of smoking upon coronary artery disease is still uncertain, it is wiser for the patient to abstain, if he can do so without causing too much distress.

Alcohol is allowed in moderation.

The patient should rest in bed for at least one month after the attack, and avoid heavy physical activity for the remainder of his life.

If his occupation does not entail great physical or mental strain, normal duties may be resumed after three months provided that the pace is slowed. If, however, congestive cardiac failure or severe angina develops, all activities must be drastically reduced.

We all await with interest the results of the work being done on lipid metabolism, and the effects of the oestrogenic hormone upon it.

The old adage that a man is as old as his arteries still stands—but for how long?

WILFRED EVANS,
Sydney.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on August 12, 1953, at the Royal Children's Hospital, Melbourne.

Bronchogenic Cyst in Infancy Causing Respiratory Obstruction.

DR. HOWARD WILLIAMS described the clinical history of a male infant, aged five months, who was found to have a bronchogenic cyst causing respiratory obstruction.

DR. RUSSELL HOWARD described the surgical details and the appearances on thoracotomy.

This case is being reported separately in another issue of the journal.

Congenital Cystic Lung with "Tension" Cysts and Respiratory Failure.

Dr. Williams then showed a male infant, aged ten days, who had been admitted to the Royal Children's Hospital on May 17, 1953, with a history of rapid breathing since birth, and of cyanotic attacks and distressed laboured respiration of two days' duration. He was the third child; the pregnancy had been normal and had gone to full term, but the labour had been rather rapid. After delivery the child cried well and had a good colour, but on the next day the respirations were observed to be rapid. This tachypnoea gradually became worse, so that by the eighth day of life the breathing was very laboured, the colour was cyanosed and the infant could suck and swallow only with considerable difficulty. The condition grew worse over the next two days.

Examination of the child on the tenth day revealed him to be severely cyanosed; he had a normal temperature and respiratory rate of 80 per minute. The chest wall was bulging anteriorly, and the costal cartilages and supra-sternal and supraclavicular spaces retracted during inspiration. The liver edge was felt two fingers' breadth below the right costal margin and the spleen one finger's breadth below the left costal margin. The left side of the chest was hyper-resonant on percussion and the air entry was very diminished. The position of the heart could not be determined accurately, but pulsation was felt to the right of the sternum, and the heart sounds were best heard over this area. Over the right side of the chest the air entry was good, but many crepitations were audible. A clinical diagnosis of left tension pneumothorax or left diaphragmatic hernia was made. The report on the radiological examination of the chest and abdomen by Dr. Piarre was as follows:

There is gross ballooning of the whole of the left lung, with right lung and heart compressed into the right hemithorax. Both sides of the diaphragm are depressed with downward displacement of the liver and spleen. The left lung has a cystic appearance with numerous loculi, and is very suggestive of bowel herniation, but no fluid levels are seen in the films taken with the child in the erect position. Barium outlines the stomach, small bowel and colon as far proximally as the transverse colon, and these structures are in the normal position. The lateral films do not give any indication of bowel passing up through the diaphragm. The appearances favour cystic lung rather than diaphragmatic hernia. No pneumothorax or mediastinal emphysema seen.

A diagnosis of multiple tension air cysts of the left lung with gross compression/atelectasis of the right lung was made. After discussion with Dr. Russell Howard it was decided to attempt to relieve the respiratory distress by needling the largest of these cysts through the seventh left intercostal space in the posterior axillary line. The initial pressure readings were +8 and +12 centimetres of water, and after the removal of 68 cubic centimetres of air the pressures read -2 and +2 centimetres. As air was continually expelled from the needle, it was left in position with an underwater seal. After this procedure the infant's colour improved, but the respirations still remained rapid. Eight hours later air ceased to bubble out under the water, the infant became very cyanosed and death appeared imminent. Two further cysts were needled and air under tension was released. Again the infant's colour slowly improved; but the respirations were still very rapid and laboured, and it became evident that the baby was almost exhausted. Surgical treatment held out the only slender hope.

Twenty-two hours after the baby's admission to hospital, Dr. Russell Howard performed left thoracotomy, during which procedure the heart stopped beating. Delivery through the wound of the grossly distended cystic lower lobe of the left lung resulted in recommencement of the heart beat. Lobectomy was performed and the chest was closed with intercostal drainage. Artificial respiration through an intratracheal tube was necessary for approximately six hours, after which time spontaneous breathing occurred and the baby's colour remained good. Several cyanotic attacks with cessation of respiration caused concern over the next two days, but then recovery was uninterrupted. The baby was discharged home three weeks after his admission to hospital, breast fed and with good air entry over the left side of the chest. Radiological examination revealed that the upper lobe of the left lung had reexpanded, but a single air-containing cyst was still present. The baby remained well until he was ten weeks old, when he contracted a cold from other members of the family and the upper lobe of the left lung became infected and partially collapsed. Treatment with aureomycin and making the baby cry resulted in some resolution, but it was too early to state what the outcome would be.

DR. ALAN WILLIAMS said that the lower lobe of the left lung was larger than normal, and was comprised largely of rounded, air-containing cysts of varying size, the largest being four centimetres, the smallest two millimetres in diameter. The cysts all had a smooth lining, and in a number communication with the bronchial tree could be demonstrated. The segmental bronchi appeared to divide normally. Air-containing lung tissue was limited to the dorsal lobe and to the anterior portion of the anterior segment. Histological examination of several sections revealed that the cyst walls were comprised of an inner layer of pseudostratified ciliated columnar epithelium, or simple columnar or cuboidal epithelium, outside which was a layer of connective tissue and smooth muscle of varying thickness. The bronchi and blood vessels were normal, while in the lung tissue between some of the cysts were seen areas of emphysema, collapse and haemorrhage. The specimen was considered to be a typical example of congenital cystic disease of the lung.

Dr. Howard Williams said that the patient illustrated the importance of accurate diagnosis of the cause of respiratory distress and cyanosis in the neonatal period. Radiological examination was essential to establish the diagnosis, and it was not justifiable to manage babies with such symptoms without proper investigation in the hope that they would recover. In retrospect, the baby would have been better operated upon as soon as the diagnosis had been made, rather than that attempts should be made to relieve the respiratory distress by needling the cysts. A review of the literature revealed that in 1925 Koontz had reported the case of a twelve-day-old infant who had died of congenital cystic

disease of the lung. Four other patients with similar clinical features to those of the patient reported had successfully undergone surgical treatment. Fischer had reported the case of a baby aged one month, Gross that of a baby aged three weeks, Burnett and Caswell that of a baby aged fifteen days, and Potts that of a baby aged five days.

DR. KATE CAMPBELL inquired about the state of the remaining lung tissue.

Dr. Howard Williams, in reply, said that the right lung appeared normal on clinical and radiological examination. A small cyst seen in the upper lobe of the left lung on radiological examination might have been a small emphysematous bleb.

Cerebellar Abscess.

DR. T. G. MADDISON presented the clinical history of a girl, aged seven years, with a right cerebellar abscess. He said that the reasons for presenting the case were, firstly, to demonstrate the unusual course of the child's illness, and secondly, to emphasize the difficulties of diagnosing brain abscess. Three and a half weeks before her admission to hospital, the child had developed earache, which persisted, and one week later she was examined by her local doctor because of drowsiness, vomiting and bilateral suppurating otorrhoea. Penicillin injections were given daily for three days, and the discharge cleared over the following week. One week before her admission to hospital she returned to school, but increasingly severe vomiting developed. Two days before her admission to hospital she complained of frontal headache, became drowsy and lost her appetite.

On her admission to hospital she was afebrile and drowsy and resented interference. She had slight neck stiffness, and both ear drums looked dull but were not injected. Generalized hypotonia and hypoaactive tendon reflexes were present. There was no nystagmus, and examination of the fundi oculorum revealed well-outlined optic disks with good physiological cups. No other abnormality was detected. Lumbar puncture produced cerebro-spinal fluid under a pressure of 120 millimetres of water. The fluid contained 12 lymphocytes and 30 polymorphonuclear leucocytes per cubic millimetre; the protein concentration was 90 milligrammes per centum and the sugar concentration was 40 to 50 milligrammes per centum. The haemoglobin value was 13.5 grammes per centum, and the white cells numbered 13,600 per cubic millimetre; 73% were polymorphonuclear leucocytes and 26% were lymphocytes. The Mantoux test, examination of the urine and radiographic examination of the skull all gave normal results. At this stage it was thought that the most likely diagnosis was meningoencephalitis; but because of the past history of otitis media and the possibility of a brain abscess no chemotherapy was given. For the next six days in hospital the patient had a slightly elevated temperature, never above 99.5° F. in the mouth. She continued to vomit twice or thrice daily and complained of headache. Her condition gradually improved, and one week after her admission to hospital she was afebrile, alert and active, and had ceased vomiting. The hypotonia was not obvious on examination. A further lumbar puncture produced cerebro-spinal fluid containing five lymphocytes per cubic millimetre and with a protein concentration of 30 milligrammes per centum. She remained afebrile, but her symptoms recurred; she began to vomit three to four times per day, complained of headache and became drowsy and apathetic. On the twelfth day, examination revealed generalized hypotonia with absence of tendon reflexes. Nystagmus was present when she looked to the right. Fundal examination revealed optic disks with physiological cups, but the nasal margins were not so well defined as previously. In view of these findings and her course in hospital, it was thought that the most likely diagnosis was a cerebellar abscess. She was examined by Dr. R. S. Hooper; but before treatment could be instituted she had a convulsive seizure. A ventriculographic examination was performed, and this revealed dilated ventricles and deviation of the aqueduct of Sylvius and fourth ventricle to the left. At operation a right cerebellar abscess was found and 15 millilitres of thick green pus were aspirated. Pneumococci were grown from this pus. After aspiration of the abscess she made an uneventful recovery.

Dr. Maddison then reviewed cases of cerebral abscess at the Royal Children's Hospital, Melbourne, over a five-year period from 1948 to 1952 inclusive. He said that over that period there had been 13 proved cases of brain abscess with five deaths. There were only two cases of cerebellar abscess in this series. Dr. Maddison said that an interesting feature was that four of the patients had presented with purulent meningitis in hospital. The cerebro-spinal fluid

ranged from normal to frankly purulent; the protein concentration of the fluid was never above 90 milligrammes per centum on the patient's admission to hospital. Dr. Maddison then briefly described details of the two patients who had suffered from cerebellar abscess.

The first patient was a girl, aged nearly five years, who had a history of left earache of two weeks' duration, associated with headache and occasional vomiting. She developed some unsteadiness of gait and dizziness on the day prior to her admission to hospital. Examination revealed her to be cooperative and afebrile, with a reddened left ear drum, slight nystagmus on looking to the left and normal fundi oculorum. She was unable to sit up, as this made her dizzy. Lumbar puncture produced cerebro-spinal fluid at a normal pressure and containing three lymphocytes per cubic millimetre. The protein concentration was 25 milligrammes per centum. A myringotomy one week after her admission to hospital revealed no fluid or pus in the middle ear, and a diagnosis of labyrinthitis was made. Her condition slowly improved over the following ten days with the aid of "Distaquaine Penicillin", and she was discharged from hospital. She was readmitted two days later because of a recurrence of the headache and unsteady gait. There was little to find on examination, but over the following three days she developed blurring of the optic disks with retinal hemorrhages and definite signs of a left-sided cerebellar lesion. Dr. Hooper drained a left-sided cerebellar abscess and she made an excellent recovery.

The second patient was a girl, aged seven and a half years, who had had a severe upper respiratory tract infection one month before her admission to hospital. This lasted for five days, and she was well for a further nine days; but then she developed intermittent headache and vomiting with some mental confusion and drowsiness. Over the last two days these symptoms had become more severe, and she complained of neck stiffness.

On examination of the patient, her temperature was 98.2° F. She was stuporose and non-cooperative. Neck stiffness was present, and she had hypotonia with weak tendon reflexes, more pronounced on the left side. Nystagmus was present on looking to the right, and she was apparently unable to look to the left. The fundi oculorum were normal. Lumbar puncture produced opalescent cerebro-spinal fluid containing 980 polymorphonuclear leucocytes and 540 lymphocytes per cubic millimetre. The protein concentration was 70 milligrammes per centum and the sugar concentration 30 to 40 milligrammes per centum, and no organisms were present in a smear. The pressure was 150 to 200 millimetres of water. She was given the usual chemotherapy for purulent meningitis, but next morning she was more stuporose. Lumbar puncture revealed the cerebro-spinal fluid to be under a pressure of 300 millimetres of water. Half an hour after the lumbar puncture she suddenly collapsed. She was resuscitated, and emergency posterior burr holes were made, but needling of the cerebellum produced no pus. She died soon afterwards. Necropsy revealed an abscess in the left lobe of the cerebellum; β -hemolytic streptococci were isolated from the pus. The left middle ear cavity contained yellow mucopus.

Dr. Maddison said that four phases were found in the classical clinical course of brain abscess. The initial phase was the phase of invasion of the brain with associated encephalitis. The quiescent phase followed. In this phase the abscess cavity was forming and the capsule was developing. In the manifest phase there was increasing pressure within the capsule, or rupture of the capsule with extension of the pus into the surrounding tissues. There were usually evidence of infection, increased intracranial pressure and focal signs. The terminal phase was characterized by meningitis and respiratory failure. However, that classical pattern was all too infrequent, and a cerebral abscess might present in any of the four phases. Dr. Maddison said that the child whose case history he had described illustrated the first three of those phases. The most important clinical features of cerebellar abscess were the following: a history of some preceding infection, usually otitis media; general signs of infection, such as fever and leucocytosis; signs of a focal cerebellar lesion—hypotonia, cerebellar ataxia, nystagmus, suboccipital tenderness and headache; signs of increased intracranial pressure; changes in the spinal fluid. Any of these diagnostic features might be absent when the child was first examined. In many cases it was only by observation and the use of surgical diagnostic procedures such as air ventriculography and exploratory brain puncture with a hollow needle that the diagnosis could be made and surgical treatment instituted.

Dr. S. W. WILLIAMS said that *otitis media* was often inadequately treated with short courses of sulphonamides or penicillin, and that might be responsible for the development of cerebral abscesses in some cases. Cerebellar abscess was difficult to diagnose, and had to be distinguished from cerebellar encephalitis resulting from virus infections such as measles.

Dr. Maddison said that for every case of cerebral abscess the diagnosis was wrongly made in four or five others.

Dr. ROBERT SOUTHEY stressed the importance of persistent headache as a symptom in small children. He said that severe persistent headache should make one suspect an intracranial lesion.

Dr. M. L. POWELL agreed with Dr. Southby, and said that headache and vomiting occurring in the early part of the day should be considered as due to space-occupying lesions until proved otherwise. It was rare to find normal cerebrospinal fluid findings in patients with cerebral abscess. Papilloedema was a late physical sign.

Dr. JOHN COLEBATCH asked if estimation of the cerebrospinal fluid globulin concentration was of any value when the total protein concentration was equivocal.

Dr. Maddison said that he was unable to answer Dr. Colebatch's question. No differential estimation of the protein in the cerebrospinal fluid had been made. It was notable that the protein concentration was only moderately raised in most cases, and some patients had normal protein concentrations.

Dr. J. W. PERRY said that he was surprised that the protein concentrations of cerebrospinal fluid were so low. Nonne-Appelt and Pandey tests for globulin probably were of little additional value.

The Role of ABO Factors in Causing Erythroblastosis Fetalis.

Dr. ELIZABETH TURNER said that since Landsteiner's discovery of the Rh factor, its role in producing *erythroblastosis fetalis* had become well known, but only recently had it been realized that similar and perhaps more disastrous damage could arise as the result of maternal sensitization to ABO factors. To quote Dr. Rachel Jakobowicz, the ABO blood group system was "conservative and tidy, simple in nomenclature, and single-minded in its antigen-antibody relationship", contrasting favourably with the "more flamboyant newer blood factors, which dominated the picture of blood group work with all the irritating liveliness of youth". The ABO system was the only one in which antibodies developed regularly as a normal phenomenon, so that it would appear that they were genetically determined. Group specific A substance was so ubiquitous in nature that immune anti-A antibodies might arise following, for example, an attack of pneumonia, an injection of T.A.B. vaccine or an injection of horse serum. Also they might arise as the result of fetal stimulation in an ABO heterospecific pregnancy—in other words, a pregnancy in which the fetus possessed an antigen inherited from its father which was lacking in its mother. Any of these external stimuli might cause the sensitization or isoimmunization of the mother with an increase or alteration of these antibodies, so that they might be strong enough to cause *erythroblastosis fetalis*.

Dr. Turner went on to say that it was not the naturally occurring α and β complete (or saline) agglutinins which were responsible for the damage, as they were of large molecular volume and rarely if ever passed through the placental barrier. The incomplete, albumin (or blocking) agglutinins, which were of smaller molecular volume, were able to pass through the placenta and produce sensitization in the mother. It was the pre-confinement alteration in titre of these antibodies which suggested the possibility of hemolytic disease occurring in the infant, and when such an alteration was detected preparations should be made to deal with an erythroblastic infant as soon as it was born. There was no known method of preventing maternal antibody formation, and while the fetus was still *in utero* it appeared to be comparatively protected from the disastrous effects of an antigen-antibody reaction. Soon after birth these effects began to manifest themselves in most tissues of the body, and an attempt should be made to lessen the force of the reaction with exchange transfusion, ACTH, cortisone, hydration and other less spectacular measures. In 22% of all marriages an ABO incompatibility was present; but, just as in the case of the Rh factor, only a small number of these mothers became sensitized and developed the potentially dangerous antibodies.

Dr. Turner said that at the Queen Victoria Hospital a sample of blood was collected from each woman at her first ante-natal visit and forwarded to Dr. Jakobowicz, and until a few months previously, when the present investigation originated, the specimens were subjected to routine testing for blood group and Rh status and for the presence of Rh-immune antibodies only. A sample of cord blood at the time of birth was also collected. By these methods, it had been possible to detect 61 women over the past year who had given birth to children with blood groups incompatible with those of their mothers. The maternal sera were then tested for the presence of incomplete anti-A and anti-B agglutinins, and the serum sample previously obtained at the woman's first visit to the ante-natal clinic was also reinvestigated for their presence. Incomplete α and β agglutinins were present during and after pregnancy in 15 of these women. In eight of these there was a low titre (1:2 or 1:4), and seven had a titre of 1:10 or higher. Of the mothers constituting the latter group, five had children with symptoms attributable to maternal-fetal blood group antagonism. The children of all the other 46 women exhibited no evidence of *erythroblastosis fetalis*. Dr. Turner then demonstrated briefly with the aid of a projected table the clinical details of the seven babies whose mothers had a titre of 1:10 or higher. As an example she quoted the clinical details of a baby who was jaundiced in the first twenty-four hours of life, with a hemoglobin value of 18.8 grammes *per centum*, an enlarged liver and a palpable spleen. The result of a Coombs test was negative, and the condition was not recognized as *icterus gravis* until, at the age of three weeks, spasms of head retraction occurred and immune antibodies were demonstrated in the maternal serum.

Dr. Turner said that it was obvious from these results that some method of earlier detection of these cases must be devised. The direct Coombs test on cord blood, which had proved such a useful indicator of hemolysis due to the Rh factor, almost invariably produced a negative result in hemolytic disease due to ABO sensitization. That might be due to the presence of group-specific A and B substances in approximately 85% of all babies (babies known as secretors). These substances would neutralize the antagonistic maternal antibodies before they could reach the fetal red blood cells. That might also explain the relatively rare occurrence of hemolytic anemia in these infants, and the fact that often in spite of jaundice there was no splenic enlargement, whilst the jaundice seemed to be more of the hepatic than the hemolytic type.

At birth the most useful single sign of hemolytic disease in these cases was the early appearance of jaundice, often accompanied by a relatively high hemoglobin level, and it was in these circumstances that kernicterus was particularly liable to occur. Wiener had stated that kernicterus was more frequent in *erythroblastosis fetalis* due to ABO sensitization than in that due to sensitization to the Rh factor. Dr. Turner said that in order to aid the detection of these cases before birth, Dr. Jakobowicz had decided to subject all sera collected at the first ante-natal visit to a test for the presence of iso-hemolysins. These could be detected in fresh sera and indicated sensitization of some variety. If iso-hemolysins were found, the presence of incomplete iso-agglutinins to the ABO system was sought, and if they were detected, monthly specimens of blood from these women were submitted for examination to determine whether any alteration in the titre of these antibodies suggestive of fetal stimulation had occurred. Some women had extremely high antibody titres even before their first pregnancies, and hemolytic disease tended to occur more often in first pregnancies in the ABO system than in first pregnancies in which sensitization to the Rh factor had occurred, except when mothers had become sensitized by previous transfusions. From the time when the investigation had been initiated at Queen Victoria Hospital until August 1, 1953, sera of 1876 women had been tested and immune antibodies strong enough to warrant further investigation had been found in 90 of these (4.8%); 89 were in group O mothers and one was in a group B mother. Only 20 of the 90 women had been delivered; but of the 20 infants, one developed mild jaundice at the age of twenty-three hours and two developed early jaundice and received exchange transfusions.

Dr. Turner, in conclusion, said that the results of the investigation were largely due to the enthusiastic support and guidance of an expert serologist in the person of Dr. Rachel Jakobowicz, to whom she owed a debt of gratitude.

Dr. RACHEL JAKOBOWICZ, in opening the discussion, said that in complications arising from maternal Rh sensitization, the laboratory could be of help by giving warning before

confinement when Rh antibodies developed or increased during pregnancy, and after the child was born, by detecting the attachment of these antibodies to the child's red cells, by a positive response to the Coombs test. Unfortunately the position was different in cases of doubtful ABO sensitization. The presence of immune anti-A and anti-B agglutinins during pregnancy did not necessarily indicate maternal sensitization, as A antigen, in particular, was ubiquitous in nature, and could lead to the production of anti-A agglutinins at any stage of life. The Coombs test on the cord blood usually produced a negative result, although the incomplete anti-A or anti-B antibodies apparently passed the placental barrier, as in the cases of group O mothers—whose sera contained, for example, incomplete anti-A agglutinins, and who had group O children—these antibodies could be detected in the cord serum at a titre corresponding to that of the maternal serum. If the baby possessed the antigen corresponding to that of the maternal immune antibody in its red cells and body fluids, this antibody could not be detected in the cord serum, as it had most likely been absorbed by the group-specific substances present in the body fluids. It was therefore left almost entirely to the clinician to make the diagnosis of fetal damage due to ABO incompatibility between mother and child. The serological tests could only support this diagnosis, or if there were no incomplete antibodies in the maternal serum corresponding to the child's antigen, possibly exclude it.

DR. J. W. PERRY asked when it was reasonable to suspect ABO sensitization in an infant with jaundice or hæmolytic, if the Coombs test result was negative, and whether serologists eliminated all other Rh types before considering ABO sensitization. Dr. Jakobowicz, in reply, said that if the Coombs test result was negative, an anti-Rh factor could be excluded, and an ABO incompatibility should be suspected. However, it was impossible to be quite certain of this.

DR. JOHN COLEBATCH said that kernicterus could occur in the absence of erythroblastosis, especially in premature infants. A certain percentage of cases was probably not due to ABO incompatibility.

Dr. Turner, in reply, said that in many cases it was impossible to say that ABO incompatibility was not to blame. Kernicterus occurring in premature infants might be due to a variety of causes; but if full-term infants with kernicterus of obscure origin were investigated in detail, it would be found that a large proportion had ABO incompatibility.

DR. L. PHILLIPS asked when it was possible to detect the presence of jaundice in infants with ABO incompatibility and giving a negative response to the Coombs test. He wondered whether the condition could be confused with physiological jaundice.

Dr. Turner, in reply, said that jaundice usually appeared in the first twelve hours of life, and this fact should distinguish the condition from physiological jaundice.

DR. M. L. POWELL asked whether the blood films differed from those found in infants suffering from the effects of Rh incompatibility.

Dr. Turner, in reply, said that there was no difference. High erythroblast counts were common.

MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Theatre, New Medical School, Frome Road, Adelaide, on October 2, 1953.

Bird Serology.

DR. J. A. R. MILES, in a contribution on bird serology, said that it had been shown that some species of bird rarely or never gave a positive reaction in the complement-fixation test, but that their serum might be capable of a combination with antigen which would render that antigen unavailable for a later reaction with a complement-fixing serum. Such serum could inhibit complement-fixation. It had also been shown that some pigeon sera could fix complement, and that others only inhibited complement-fixation.

When pigeons were infected with Murray Valley encephalitis, some birds produced only inhibiting antibodies, but others first inhibited complement-fixation, then fixed and later returned to inhibiting before finally all reaction dis-

appeared. After reinoculation the same series of reactions was again seen.

When sera giving the two main types of different reaction were mixed, the various intermediate reactions could be stimulated; this suggested that the two effects were in the main due to two types of antibody, both of which might be produced by the same bird under suitable conditions. A similar phenomenon had been observed in some other Columbiformes and might occur in a number of other groups of birds.

The Thyroid Gland and the Lower Chordates.

MR. I. THOMAS said that the thyroid gland arose ontogenetically as an outgrowth of the floor of the mouth. Its ability to accumulate iodine developed relatively early in forms which had a free-living larval life, such as tadpoles and cyclostomes, but in mammals, for example, the rat, the ability was not acquired until shortly before parturition.

In phylogeny, the gland could be traced back with some certainty to the Cyclostomata. In the adult lamprey (*Entosphenus lamottii*), the thyroid gland consisted of a number of isolated follicles, representing its most primitive anatomical condition. Embryologically it was derived from certain cell elements of the subpharyngeal gland of the ammocete larva of the cyclostome, and it had been shown by work with radioactive tracers that those cells could accumulate iodine. The work had been confirmed in the ammocete larva of the Australian lamprey, *Geotria australis*. Little was known of the function of the thyroid or of the subpharyngeal gland in cyclostomes, though it was possible that the latter was concerned with the capture of the minutely particulate food of the larva. Mammalian thyroid did not accelerate the metamorphosis of ammocete to adult, nor did the administration of inorganic iodine. Neither would the adult cyclostome thyroid promote metamorphosis in amphibian tadpoles.

The subpharyngeal gland was, morphologically, very similar to the endostyle of the lower chordates, for example, Amphioxus and the tunicates. No demonstrable uptake of radioactive iodine has been shown in the endostyle of Amphioxus or of three tunicate genera (*Perophora*, *Styela* and *Ciona*) which had been investigated. In *Perophora*, however, iodine uptake had been observed in certain tissues of the stolon which were derived from the pharyngeal region.

In some Hemichordata, a food groove in the base of the branchial region of the alimentary canal bore some morphological and functional resemblance to the endostyle of tunicates and Amphioxus. No active uptake of radioactive iodine had been demonstrated by the cells of that groove, though there were indications of its accumulation in two longitudinal muscle bands which lay immediately underneath it. Those muscles had no direct functional connexion with the alimentary canal, and it was difficult to explain why those tissues should selectively take up the element.

Variation in ILT Virus.

MR. M. PULSFORD read a paper on variation in ILT virus.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A CHANGE OF PROFESSION FOR TWO MEDICAL PRACTITIONERS.¹

[The Australian Medical Journal, May, 1863.]

A CORRESPONDENT of the Port Denison Times, writing from Burketown, says that two medical men who went there in the hope of making a fortune out of the large practice to be got from the constantly prevailing endemic fever have abandoned the profession and are now respectively keeping a public house and tending a mob of "Kanakas" whatever these last may be.

¹ From the original in the Mitchell Library, Sydney.

Obituary.

ARCHIBALD CRAIG TELFER.

We are indebted to Dr. Richmond Jeremy and Dr. Errol Maffery for the following appreciation of the late Dr. Archibald Craig Telfer.

Dr. Archibald Craig Telfer was born at Sydney and died there on February 18, 1953, a few days before his fiftieth birthday. He was educated at Tamworth and Fort Street High Schools and entered the University of Sydney in 1922 with a public exhibition. His first three undergraduate years were spent in residence at Wesley College, which he represented in intercollegiate tennis. He graduated in medicine in 1927 and was appointed to the staff of Sydney Hospital, where he served as junior and senior resident medical officer and as resident pathologist. From March, 1931, he was assistant medical superintendent until appointed medical superintendent in 1934. In 1933 he was granted leave of absence from his hospital duties in order to visit the United Kingdom and Europe, and whilst there he carried out post-graduate studies at Edinburgh, and made observations of overseas methods of hospital administration, which were



embodied in a written report on his return to Sydney Hospital (it was always a source of conjecture to him whether anybody ever read that report). After leaving Sydney Hospital in 1937, he visited the United States of America for the purpose of furthering his studies in urology. On his return from America he commenced practice in Macquarie Street, and was appointed to the honorary staff of Sydney Hospital in the young and vigorous department of urology, to which he devoted much of his plentiful enthusiasm and inspiration. He was also honorary urologist to the Balmain District Hospital and consulting urologist to the Women's Hospital, Crown Street.

Early in his medical career Dr. Telfer became interested in medical social work, an interest which was greatly stimulated during his visit to the United States of America. On his return to Sydney he was instrumental in establishing an almoner's department at Sydney Hospital, and played an active part in the formation of the New South Wales Institute of Hospital Almoners. He was a member of the council from its inception, and at the time of his death was

vice-president of the institute and vice-chairman of its council. His practical interest in medical social work was well demonstrated by the extent to which he called upon almoners to help his patients.

Dr. Telfer was a member of the British Association of Urological Surgeons and of the International Urological Society. He was a foundation member of the Urological Society of Australasia and served on its executive council from its inception until his death. He was vice-president of the society in 1950, president in 1951, and programme arranger for its annual clinical meeting from 1952. During his tenure of the latter office he gave considerable time to stimulating the younger urologists throughout Australia to get under way with original work for presentation in some later year.

Archie Telfer's was a strong, a dynamic personality, coupled with the highest ideals and with great tolerance—he could always see the other man's point of view, though he did not necessarily agree with him. His strength of character was very apparent to all who knew him well by the way in which he "took" the severe limitations to physical activity imposed upon him by ill health during the last fourteen years of his life. During the late nineteen twenties and early thirties he was an enthusiastic skier; he was a member and committee man of the University and the Millions Ski Clubs, representing the latter club on the Ski Council of New South Wales. He made several difficult cross-country treks, mainly in company with R. M. Gelling, and also with the late George Aalberg. He and his companions explored the Main Dividing Range in winter and in summer, their object being to know the range and to survey the existing hut accommodation, and thus to help open up our snow country to the average skier. He enjoyed the arduous way, and on these trips took delight in being forced by conditions to camp at night in the open snow. All this was denied him by illness in 1939; the tempo of his existence was completely changed. His love of literature and his interest in the flora and fauna of the Australian bush helped him to readjust his way of life. The misfortune of ill health did not produce embitterment, as might have been expected in one who had always shown such tireless energy and enthusiasm—rather did it produce a greater serenity and depth of understanding of the troubles of others. His great interest in the problems of his own branch of surgery seemed to grow as he became less able to undertake the things he wanted to do. His natural capacity for friendship and a wide knowledge of medicine and surgery made him sought after for advice about matters often far removed from the field of his own speciality. He was one of the public-spirited who voluntarily gave their time to the militia forces in preparation for war. This was another activity from which he was forced to resign. It was a bitter blow to one of his temperament that he was unable to serve in the 1939-1945 war. He leaves a widow and son, to whom the sincere sympathy of his colleagues and friends is extended.

We wish to acknowledge the help of Miss F. K. Ogilvie, M.B.E., Dr. J. E. Wiseman and Dr. Hugh Pearson in the compilation of these notes.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course for the Diploma in Diagnostic Radiology.

The Post-Graduate Committee in Medicine in the University of Sydney announces that it is proposed to conduct a course for Parts I and II of the diploma in diagnostic radiology for twelve months beginning March 22, 1954. The course of lectures is part time, but for those who have no previous experience in diagnostic radiology and who wish to qualify for the diploma, arrangements can be made for their allocation to a recognized hospital over the full-time period of twelve months.

Those interested in attending the course are asked to communicate, as soon as possible, with the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, where further particulars concerning the course and the diploma may be obtained.

Clinical Meeting at Balmoral Naval Hospital.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a clinical meeting will

be held at the Balmoral Naval Hospital on Tuesday, March 9, 1954, at 2 p.m., when Dr. Hugh Smith will speak on "Internal Derangements of the Knee Joint". Clinical cases will be shown following the lecture. All members of the medical profession are invited to attend.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

INTENDING CANDIDATES for the membership of The Royal Australasian College of Physicians are reminded that the closing date for the receipt of applications for the next examination is Saturday, March 6, 1954.

The written examination will be held in capital cities on Saturday, April 3, 1954, and the clinical examination will be held in Melbourne from approximately Friday, May 21, to Tuesday, May 25, 1954. Application forms may be obtained from the Honorary Secretary of the College, 145 Macquarie Street, Sydney.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the Medical Practitioners Act, 1938-1950, as duly qualified medical practitioners: *Deerbon, Murray Karei* Francis, M.B., B.S., 1951 (Univ. Melbourne); *Kingston, Clive* William, M.B., B.S., 1940 (Univ. Queensland); *Madigan, Michael* Rosebourne, M.B., B.S., 1952 (Univ. Adelaide); *Moysey, Colin* David Edwards, M.B., B.S., 1951 (Univ. Melbourne); *Paterson, Richard* Simpson, M.B., B.S., 1952 (Univ.

Queensland); *Pinkerton, Grace* Dorothy, M.R.C.S. (England), L.R.C.P. (London), 1952; *Troski, Samuel*, M.B., B.S., 1946 (Univ. Melbourne); D.O., 1953 (Univ. Melbourne); *Trudinger, Lawrence* Robert, M.B., B.S., 1939 (Univ. Melbourne); *Webster, Roland* Ferrah Kaye, L.R.C.P. (Edinburgh L.R.C.S. (Edinburgh), 1932, L.R.F.P.S. (Glasgow), 1932; *Grivas, Frank*, M.B., B.S., 1953 (Univ. Sydney).

The following additional qualifications have been registered: *Carey, Harvey* McKay (M.B., B.S., 1941, Univ. Sydney), D.G.O., 1948 (Univ. Sydney), M.R.C.O.G. (London), 1951, F.R.C.S. (Edinburgh), 1950; *Greenaway, Thomas* Moore (M.B., Ch.M., 1925, Univ. Sydney, M.R.C.P., 1934, London), F.R.C.P. (London), 1951; *Harvey, Henry* Peter Burnell (M.B., B.S., 1948, Univ. Sydney), M.R.A.C.P., 1953; *McDonald, John* David (M.B., B.S., 1947, Univ. Sydney), M.R.A.C.P., 1953.

The following notification is published in the *New South Wales Government Gazette*, Number 3, of January 15, 1954.

It is hereby notified for public information that the name of *Clifford Kenneth* Hemmingsway, M.B., B.S., 1942, Univ. Sydney, has been removed from the Register of Medical Practitioners for New South Wales in terms of Section 30 of the Medical Practitioners Act, 1938-1953.

Congresses.

FOURTH COMMONWEALTH HEALTH AND TUBERCULOSIS CONFERENCE.

THE fourth Commonwealth Health and Tuberculosis Conference, organized by the National Association for the Prevention of Tuberculosis, will be held in London from June 21 to 25, 1955. The programme will include lectures, discussions and clinical meetings, as well as practical demonstrations and visits to sanatoria, hospitals and clinics. Further information may be obtained from the Secretary-General, NAPT, Tavistock House North, Tavistock Square, London, W.C.1.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 30, 1954.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory. ²	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	1(1)	4
Amoebiasis
Ancylostomiasis	1	1
Anthrax
Bilharziasis
Brucellosis
Cholera
Chorea (St. Vitus)
Dengue
Diarrhoea (Infantile)	1(1)	10(9)	8(8)	1	20
Diphtheria	4(3)	4(3)	2(1)	..	7(5)	17
Dysentery (Bacillary)	11(9)	..	2(1)	13
Encephalitis	2(1)	1(1)	3
Filariasis
Homologous Serum Jaundice
Hydatid	2(1)	2
Infective Hepatitis	1(1)	10(3)	5(1)	16
Lead Poisoning
Leprosy
Leptospirosis	2	2
Malaria	7	13
Meningococcal infection	4(1)	2(2)	17
Ophthalmia	17
Ornithosis
Paratyphoid
Plague
Pollomyelitis	15(12)	5(4)	1	2(1)	7(5)	30
Puerperal Fever	11(9)	21(8)	32
Rubella	1(1)	1
Salmonella Infection	1(1)	22
Scarlet Fever	3(3)	9(6)	3(2)	6(2)
Smallpox	1	1
Tetanus	131	131
Trachoma
Trichinosis
Tuberculosis	52(43)	20(17)	32(18)	9(5)	4(3)	4(1)	121
Typhoid Fever	1(1)	1
Typhus (Flea-, Mite- and Tick-borne)	1(1)	1
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

² Figures not available.

³ Figures incomplete owing to absence of returns from Northern Territory.

Medical Appointments.

Dr. J. J. Witton Flynn has been appointed President of the New South Wales Medical Board.

Dr. E. G. MacMahon has been appointed a member of the New South Wales Medical Board.

Dr. R. J. Sargent has been appointed medical superintendent of the Northfield Wards of the Royal Adelaide Hospital.

Dr. J. S. B. Lindsay has been appointed psychiatrist of the Mental Hygiene Branch of the Department of Health, Victoria, pursuant to the provisions of Section 18 of the *Mental Hygiene Authority Act, 1950*.

Dr. Margaret Matilda Patterson has been appointed resident anaesthetist in the Hospitals Department of South Australia.

Dr. D. A. Handley has been appointed registrar in clinical pathology in the Hospitals Department of South Australia.

Dr. B. A. Connor has been appointed a public vaccinator to the Shire of Kaniva, Victoria.

Dr. D. T. Gilbert has been appointed a public vaccinator to the City of Sandringham, Victoria.

Dr. T. J. Cotter has been appointed government medical officer at Innisfail, Queensland.

Dr. Trevor Nelson Hatfield and Dr. Brian Gordon Thomas have been appointed Quarantine Officers at Fremantle and Port Lincoln respectively under the provisions of the *Quarantine Act, 1908-1950*.

Dr. H. B. Taylor and Dr. C. B. Cox have been appointed analysts within the meaning and for the purposes of the *Pure Food Act, 1908*, in the Department of Public Health, New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Mezo, Bela Albert, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1950*, District Hospital, WallSEND, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Appleby, Bruce Robert, M.B., B.S., 1954 (Univ. Sydney); Bain, John Joseph (Flight-Lieutenant), M.B., B.S., 1954 (Univ. Sydney); Campbell, Ian Alexander, M.B., B.S., 1954 (Univ. Sydney); Clevee, Noel Pitt, M.B., B.S., 1954 (Univ. Sydney); Coleman, Roger Henry, M.B., B.S., 1954 (Univ. Sydney); Cooke, Samuel Lindsay, M.B., B.S., 1954 (Univ. Sydney); Coombes, Bruce William, M.B., B.S., 1954 (Univ. Sydney); Doust, Ian Stafford, M.B., B.S., 1954 (Univ. Sydney); Fox, Mary Louise, M.B., B.S., 1954 (Univ. Sydney); Ledermann, Ruth, M.B., B.S., 1954 (Univ. Sydney); Lock, Raymond Junior, M.B., B.S., 1954 (Univ. Sydney); Mayer, Beris Olwyn, M.B., B.S., 1954 (Univ. Sydney); Nelson, David Yeates, M.B., B.S., 1954 (Univ. Sydney); Norington, Bradney William, M.B., B.S., 1954 (Univ. Sydney); Payne, William Harold, M.B., B.S., 1954 (Univ. Sydney); Pelly, Anthony D'Arcy, M.B., B.S., 1943 (Univ. Sydney); Raine, June Marion, M.B., B.S., 1954 (Univ. Sydney); Rosler, Alice Mary, M.B., B.S., 1954 (Univ. Sydney); Scott, Andrew Murray, M.B., B.S., 1954 (Univ. Sydney); Segelov, Phillip Myer, M.B., B.S., 1954 (Univ. Sydney); Skinner, Haydn French, M.B., B.S., 1954 (Univ. Sydney); Storey, Gilbert Norman Bruce, M.B., B.S., 1954 (Univ. Sydney); Sundin, Paul William, M.B., B.S., 1954 (Univ. Sydney); Tomlin, Alwynne Dorothy, M.B., B.S., 1954 (Univ. Sydney); Tooth, Maxwell John, M.B., B.S., 1954 (Univ. Sydney); Verge, John Mackay, M.B., B.S., 1954 (Univ. Sydney); Vickers, John William, M.B., B.S., 1954 (Univ. Sydney); Whish, Keith Milroy, M.B., B.S., 1954 (Univ. Sydney); Wilson, Kenneth Peter, M.B., B.S., 1954 (Univ. Sydney); Wilson, Peter Charles McLeod, M.B., B.S., 1954 (Univ. Sydney); Young, Ailsa Margaret, M.B., B.S., 1954 (Univ. Sydney); Zorbas, John, M.B., B.S., 1954 (Univ. Sydney); Bosler, John Martin, M.B., B.S., 1953 (Univ. Sydney); Coote, Barry Desmond, M.B., B.S., 1953 (Univ. Sydney); Grivas, Frank, M.B., B.S., 1953 (Univ. Sydney);

Johnson, Ross Arthur, M.B., B.S., 1953 (Univ. Sydney); Moffitt, Paul Sydney, M.B., B.S., 1953 (Univ. Sydney); Rush, Sidney James, M.B., B.S., 1953 (Univ. Sydney); Adcock, Vincent Joseph, M.B., B.S., 1952 (Univ. Sydney); Easy, John Edmund, M.B., B.S., 1952 (Univ. Sydney); Keldouls, Theo, M.B., B.S., 1952 (Univ. Sydney); Morris, Robert Barry, M.B., B.S., 1952 (Univ. Sydney); Radziowsky, Nikolay, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1950*; Van Der Poorten, David, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1950*.

Diary for the Month.

- MARCH 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
- MARCH 3.—Western Australian Branch, B.M.A.: Council Meeting.
- MARCH 5.—Queensland Branch, B.M.A.: General Meeting.
- MARCH 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

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